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Acute Hemorrhagic Pancreatitis: Experimental Surgical Procedures Applied to Alter the Course of the Disease

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THE experimental production of acute hemorrhagic pancreatitis in the laboratory animal by a standard method would serve as a baseline for comparison of the studies of different investigators. The gross pathologic changes considered basic to this disease process consist of extensive pancreatic necrosis with intrapancreatic and extrapancreatic hemorrhage, in association with diffuse, spreading peritonitis, local fat necrosis and systemic toxemia.[1] This type of pancreatic inflammation should be clearly differentiated from the milder forms of pancreatitis, such as interstitial pancreatitis (pancreatic edema), and from chronic pancreatitis. The reproduction of disease processes in the experimental animal has been applied successfully in the study of other problems: for example, the formation of peptic ulceration in the dog by drainage of the duodenum into the ileum;[2] the production of cardiac infarction in the dog by ligation of the anterior de-

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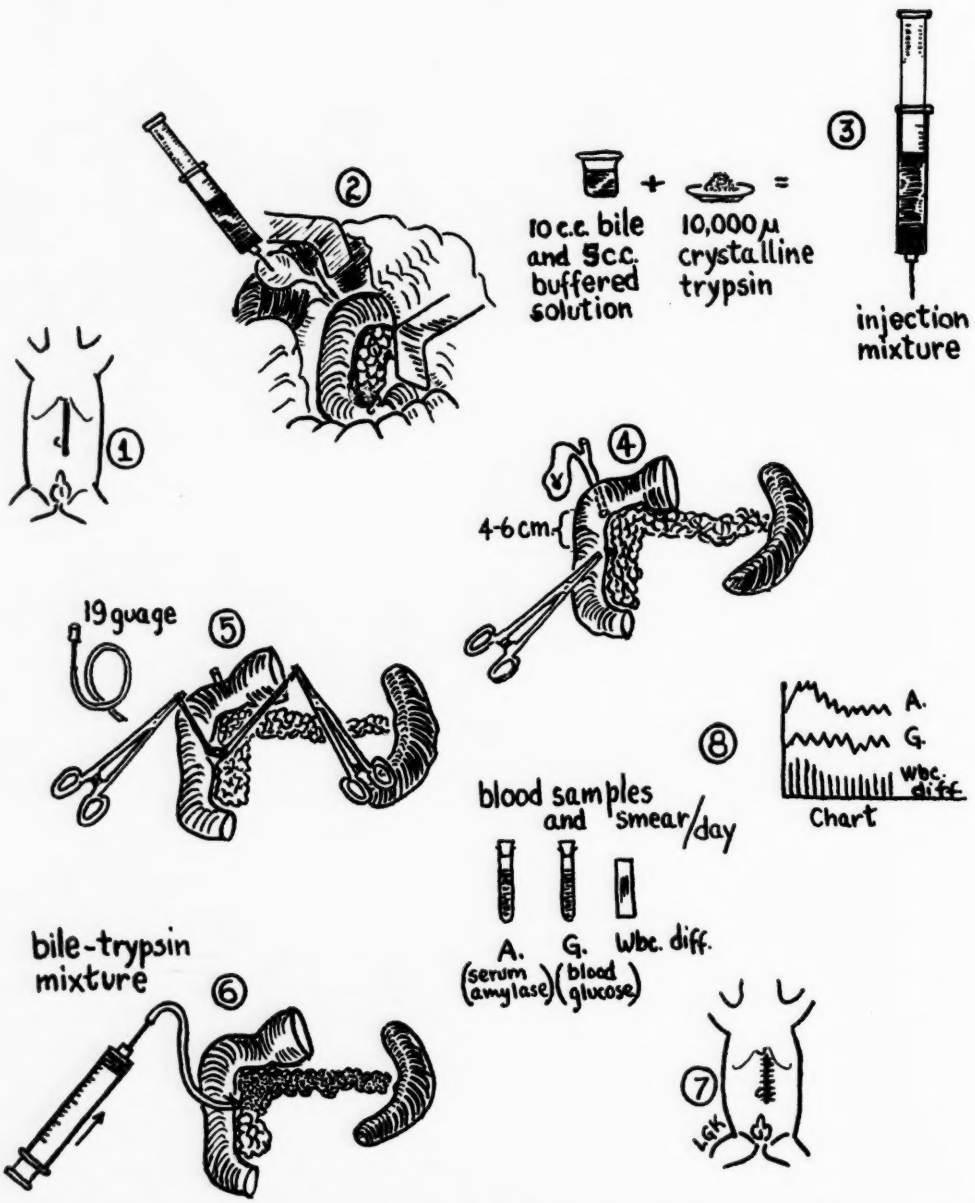

Fig. 1.—A method of producing acute hemorrhagic pancreatitis in the experimental laboratory animal (dog). *1*, midline laparotomy incision; *2*, aspiration of 10 cc. of bile from gallbladder; *3*, bile mixed with crystalline trypsin (10,000 units); *4*, "accessory" larger pancreatic duct identified; *5*, duct mobilized and polythene catheter inserted into lumen, toward body of pancreas; *6*, bile-trypsin buffered mixture injected into pancreatic duct system; *7*, abdominal incision closed by suture; *8*, data prior to and subsequent to operative procedure (blood samples obtained for serum amylase and blood sugar levels and white blood cell (Schilling) differential count).

scending branch of the left coronary artery,[3] and the establishment of arterial hypertension in the dog by clamp application to renal artery.[4] Laboratory animals afflicted with these processes have served as subjects for the application and evaluation of different therapeutic measures. It is the purpose of this report to describe (a) a method that consistently reproduces acute hemorrhagic pancreatitis in the dog; (b) the gross and microscopic changes that occur subsequent to the reproduction, and (c) the results of certain surgical procedures applied in an effort to influence the degree, extent and duration of this acute inflammatory process.

A method of producing acute hemorrhagic pancreatitis in the dog is described; no originality is claimed as to its mode of application. The method has merit in that it embodies the predominant concept of the pathogenesis of acute hemorrhagic pancreatitis; it consistently produces the same lesions each time it is applied, and it has inherent simplicity. This simplicity has led the authors to the opinion that it could serve as an experimental tool for investigators, as well as a teaching exercise for surgical resident trainees. It is suggested that the multiple and varied experimental procedures previously reported may have had a tendency to confuse and delay a long-needed concrete effort to find a definitive therapeutic approach to this disease in man.

A method which would embody an acceptable concept[5] of the pathogenesis of acute hemorrhagic pancreatitis, which could produce this acute process in an experimental animal with predictable results and which could be duplicated by other investigators would make possible a concerted application of medical and surgical procedures to alleviate this disease. During the period from 1900 to 1959, thirty-nine different experimental procedures (see *Addenda*, Note 1) have been described which reportedly produce pancreatitis in a variety of animals, exclusive of three earlier methods (Bernard,[6] 1856; Hlava,[7] 1890, and Hildebrand,[8] 1895). No single procedure has been universally adopted for the experimental production of this disease. In an acceptable approach, the procedure should cause rapidly developing hemorrhagic necrosis of the pancreas in the presence of complete ductal obstruction, enabling the exocrine outflow to be diffused into the parenchymal tissue by means of the mechanical rupture of the smaller ducts. The proposed technic attempts to mimic this sequence of events, and uses the mortality rate (approaching L.D.50) as the end point. In this manner, any agency the would influence the mortality rate in the direction of survival would indicate a significant therapeutic advance.

Four factors are considered necessary to produce this acute inflammatory process and are influenced, in part, by the structural configuration of the canine pancreatic duct system. These factors are bile, trypsin, an alkaline medium of optimum pH, and ductile rupture. The addition of trypsin to bile markedly increases the severity of the inflammation by directly activating the intrapancreatic ferments; the intraductal (pancreatic) injection of bile, alone, often leads solely to edema, unless large quantities are used to effect ductile rupture. A buffered phosphate solution (*Addenda*, Note 3) may be used as a carrier to maintain an optimum *p*H (7.1) for the action of the enzyme, trypsin.[9] Rupture of the ducts is brought about by controlling the volume

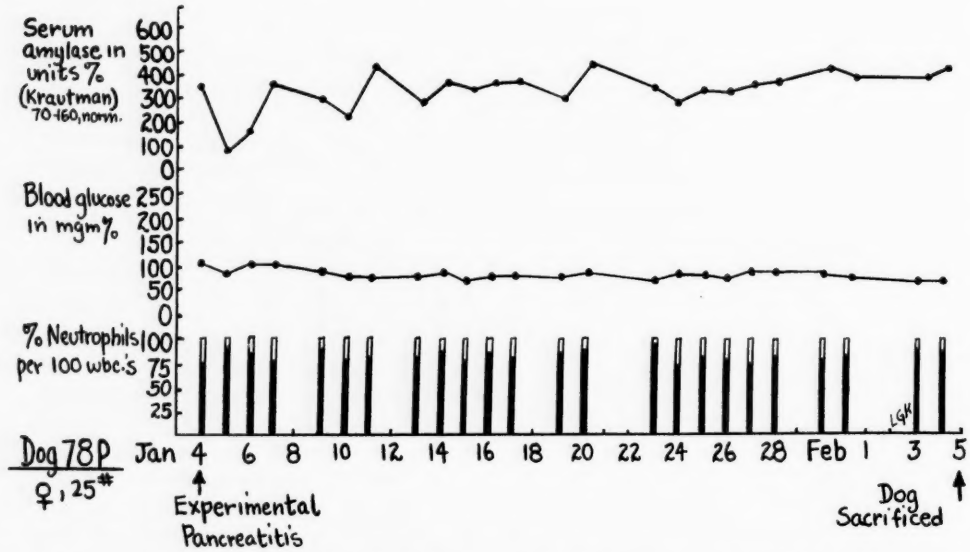

Fig. 2.—Experimental pancreatitis (described procedure). Chart of dog which survived for thirty days; serum amylase level and the leukocyte differential count (Schilling) indicate several phases of resolution and reexacerbation of acute inflammatory process.

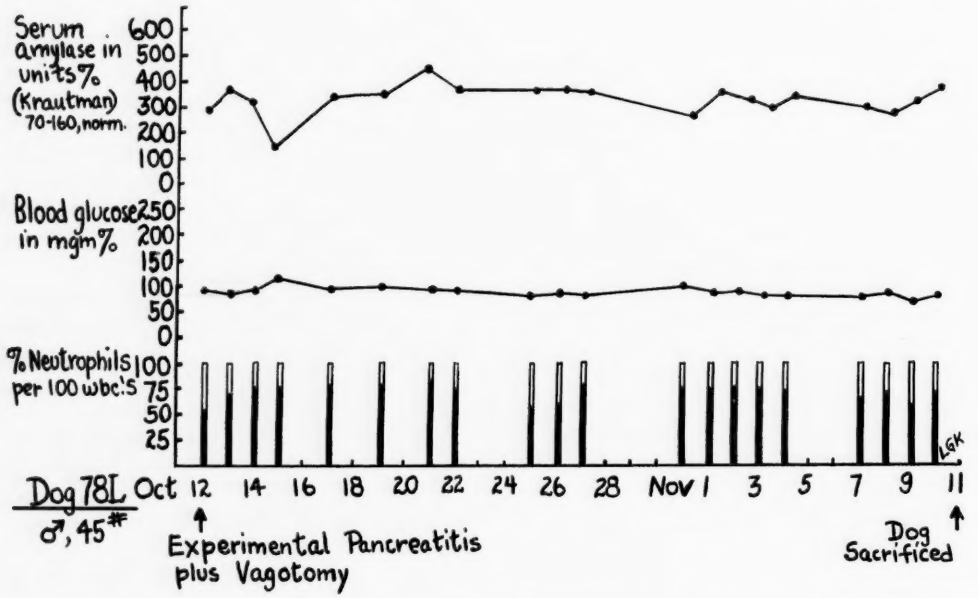

Fig. 3.—Experimental pancreatitis plus vagisection. Chart of dog which survived for thirty days. Late increase in serum amylase values (nine days after initiation of acute process) and moderate polymorphonuclearcytosis suggest period of additional pancreatic inflammation and necrosis.

of injected solution and the force used in the injection. The grossly visual diffusion of the bile-trypsin solution within the pancreatic parenchyma confirms the presence of ductile rupture and obviates the use of direct pressure measurements. Morphologically, the canine pancreas has long transverse and vertical limbs, which lie within the convexity formed by the stomach and the freely mobile duodenum. There are two duct systems within the pancreas: a smaller "main" duct, which drains the transverse limb into a common ampulla with the ductus choledochus; and a larger "accessory" duct, which drains the vertical limb and commonly a portion of the transverse limb. Within the body of the pancreas these two duct systems have communications. The smaller "main" duct may be nondemonstrable in 50 per cent of instances;[11] in addition, owing to its size, it cannot serve as a consistent decompression outlet, should the larger duct be injected with an irritant solution or be ligated. The larger "accessory" duct empties by a single outlet into the duodenum, 4 to 6 cm. distal to the bile duct ampulla.

The method used to produce acute hemorrhagic pancreatitis in the dog was as follows: The animal was anesthetized with morphine sulfate (subcutaneously, 3 mg. per kilogram of body weight) and nembutal (intraperitoneally, 30 mg. per kilogram). (Each dog weighed 12 to 20 Kg. or more; in dogs of lesser weight the pancreatic duct was observed to be too small for easy identification, and the ease of duct location is important, as surgical trauma due to excessive dis-

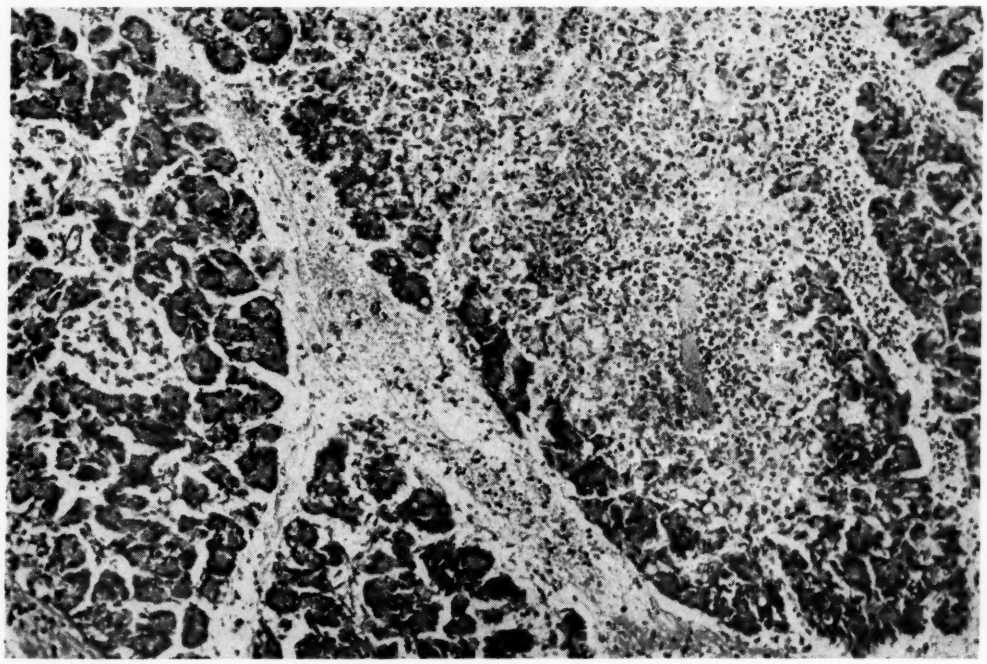

Fig. 4.—Experimental pancreatitis (described procedure). Microsection discloses inflammatory exudate replacing acinar tissue; intact acini at periphery of lobule indicate central-ductile origin of disease process produced by artificial mechanical rupture of pancreatic duct system. × 150.

section can produce local pancreatitis). Under sterile operating room technic a midline abdominal incision was made to expose the internal organs (Fig. 1). Ten cc. of bile was withdrawn from the gallbladder; a single suture closed the puncture hole in this organ. The bile was mixed with 10,000 units of crystalline trypsin (Armour's Tryptar)® (*Addenda,* Note 2) and 5 cc. of a buffered phosphate solution (*Addenda,* Note 3) in a sterile container. The larger ("accessory") pancreatic duct was carefully mobilized just external to the duodenum, in the cleft between the pancreas and the descending duodenal loop. A polyethylene catheter was threaded into the cephalic portion of the pancreatic duct, and the bile-trypsin mixture (total volume, 15 cc.) was injected by means of a syringe under moderate pressure. The catheter was removed and the duct ligated. Within a few minutes the pancreas turned nutmeg brown, and within thirty minutes a parenchymal hemorrhagic reaction could be noted. Additional surgical procedures were performed *(vide infra)* as the situation dictated. The operation was terminated by suture closure of the incision. All dogs were given routine pen care and a standard diet (horse meat and kibbled meal). Antibiotics were not used. Prior to the surgical procedure and subsequently, at almost daily intervals, tests for serum amylase (*Addenda,* Note 4), blood sugar (*Addenda,* Note 5) and a white blood cell differential (Schilling) count were made to gauge the severity of the reaction and to correlate the laboratory data with the clinical course of the disease (Figs. 2 and 3). The animals that survived were permitted to live for thirty days, after which

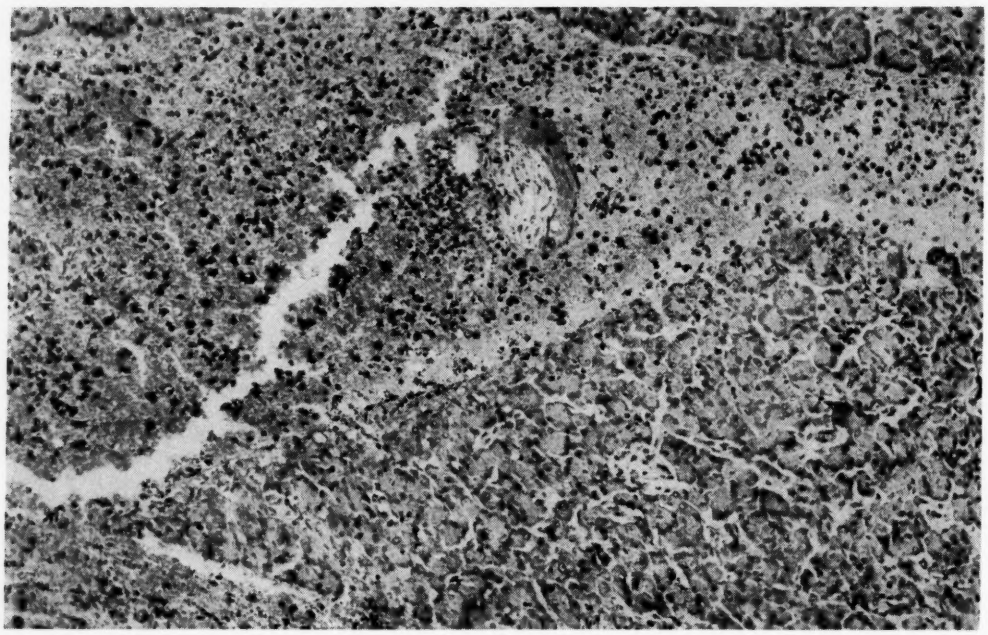

Fig. 5.—Experimental pancreatitis (described procedure). Microsection discloses interlobular pancreatic spaces filled with hemorrhagic exudate containing some pigment from hemolysed erythrocytes. × 150.

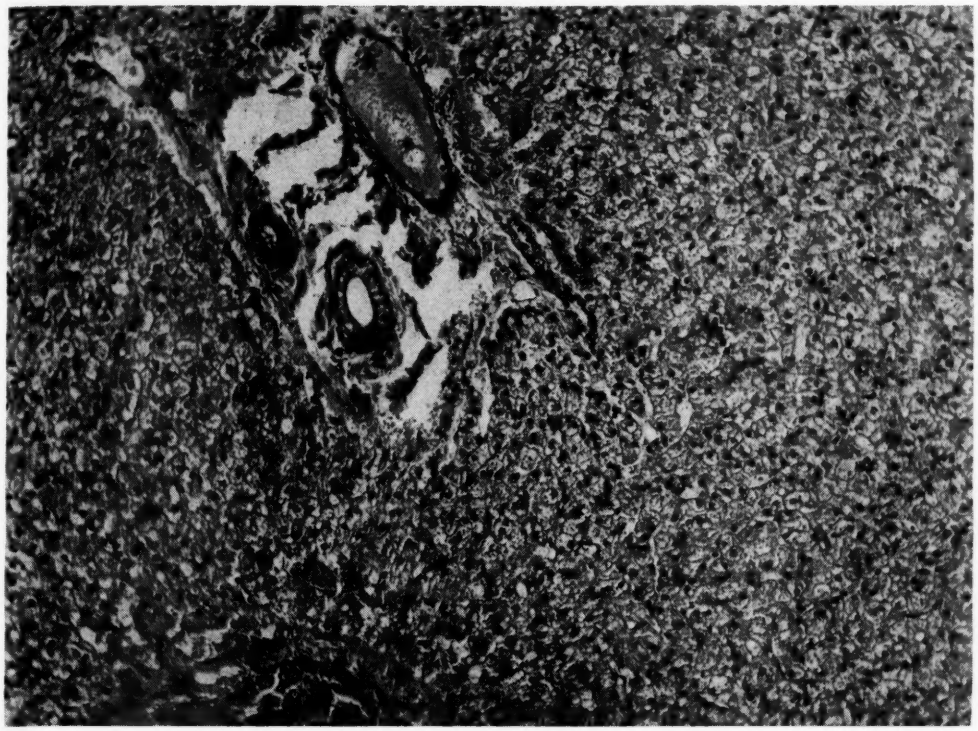

Fig. 6.—Experimental pancreatitis (described procedure). Animal that survived thirty days after initiation of acute inflammatory process; microsection of hepatic cells discloses diffuse vacuolization; portal triad structures appear at top of figure. × 150.

time they were killed. Autopsies were performed on all animals; the gross observations were noted, and microscopic sections were made of the pancreatic, hepatic, renal, splenic, and omental tissues (Figs. 4, 5, 6, 7, 8 and 9).

Forty-seven surgical procedures were performed in an effort to study and to modify the toxic and lethal effects of acute pancreatitis in the dog. It was considered that reduction of the pancreatic enzyme (especially trypsin and lipase) output, local neutralization of the pancreatic enzymes, external drainage of the enzyme output or removal of the source of these enzymes would favorably influence the course of this inflammatory process. Toward this end, division of the vagus nerve at the time of induction of pancreatitis, division of the vagus nerve thirty days prior to the induction of pancreatitis, external drainage of the pancreatic duct and resection of the inflamed portion of the pancreas were the therapeutic procedures applied. Vagisection thirty days prior to the formation of the acute pancreatic inflammation was performed in an effort to provide a pancreas with an initial low enzyme output. All divisions of the vagus nerve were subdiaphragmatic. Ductal drainage was effected by exteriorizing that portion of the pancreas which contained the larger "accessory" duct and thus provide the pancreatic secretions an external egress via an intraductal polyethylene catheter. Pancreatic resection

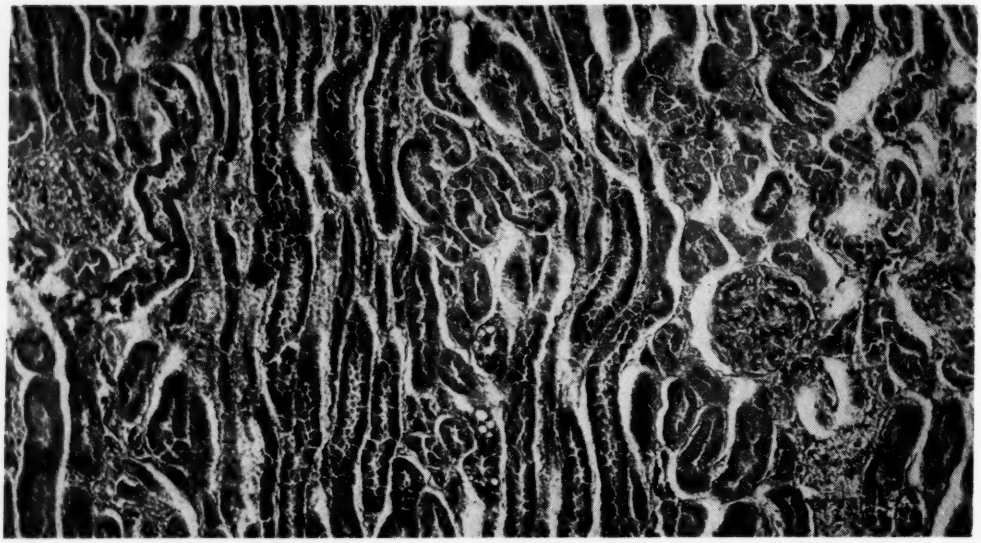

Fig. 7.—Experimental pancreatitis (described procedure). Animal did not survive. Renal tubules disclose hyaline degeneration; these changes were not consistently noted, and this particular microsection was best example of this pathologic change. Such renal damage might explain appearance of albuminuria in some instances of acute hemorrhagic pancreatitis in man. × 150.

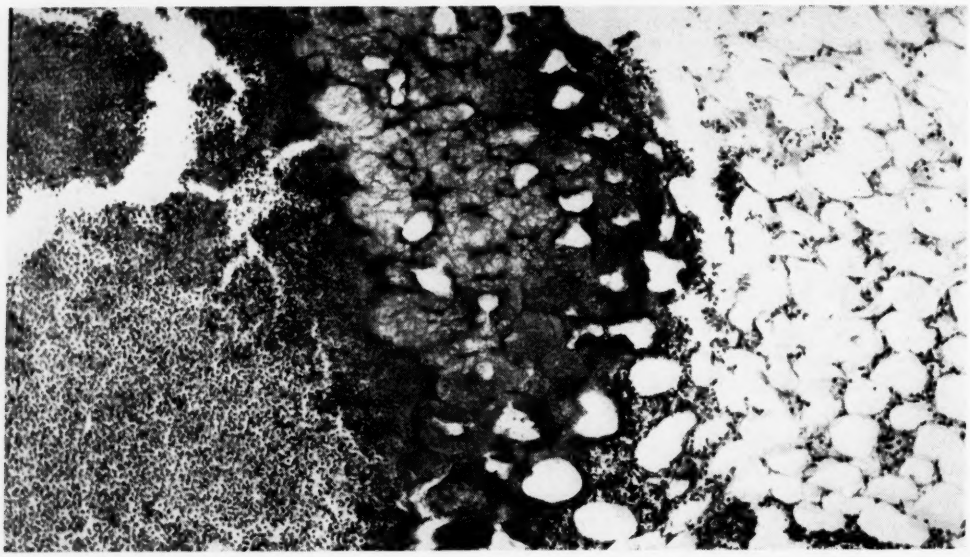

Fig. 8.—Experimental pancreatitis (described procedure). Microsection of omentum discloses normal fat cells, necrotic fat cells and inflammatory cells, as three distinct layers. × 150.

was subtotal (70 to 80 per cent) and was supplemented by intravenous infusions of Dextran® and/or 5 per cent dextrose solutions. The results (*Addenda*, Note 6) disclose that none of these "therapeutic" surgical procedures affected the

mortality rate when compared to the "baseline" group of 10 dogs (experimental pancreatitis produced as described). In the "external ductal drainage" and in the "resection" groups, it was noted, of the animals that died, that they usually had lived at least four to six days, an increase in the survival time 200 per cent over that of the "baseline" group. This indicates some amelioration of the lethal effect, although there was no improvement in the final mortality rate. Further studies are being instituted and are in progress to modify or change the technic and to discover whether the surgical approach to this disease has merit.

ADDENDA

NOTE 1.—The experimental methods used to produce pancreatitis in the laboratory animal are here listed chronologically. A few in the literature have not been included because they resemble almost identical data previously reported.

1. Injection of pancreatic duct with bile and sweet oil; dog (Bernard,[6] 1856).
2. Injection of pancreatic duct with gastric juice; dog (Hlava,[7] 1890).
3. Injection of pancreatic tissue, ground glass, and water mixture into pancreatic parenchyma; cat (Hildebrand,[8] 1895).
4. Injection of pancreatic duct with hydrochloric, nitric and chromic acids; dog (Flexner,[12] 1900).
5. Injection of pancreatic duct with bile, soaps, fatty acids, and formaldehyde; dog (Opie,[13] 1901).
6. Ligation of pancreatic duct at height of digestion; dog (Hess,[14] 1903).
7. Injection of pancreatic duct with bile salts; dog (Flexner,[15] 1906).
8. Injection of pancreatic duct with activated commercial trypsin; dog (Polya,[16] 1912).
9. Injection of pancreatic and common bile ducts with bile and fecal material; dog (Nordman,[17] 1913).
10. Injection of pancreatic duct with pig's bile; dog (Whipple and Goodpasture,[18] 1913).

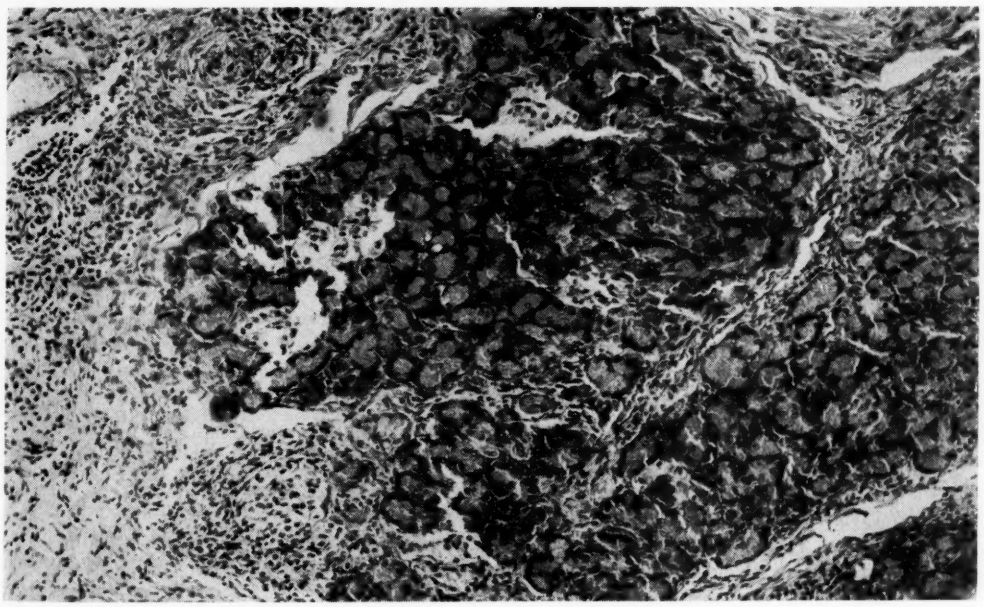

Fig. 9.—Experimental pancreatitis (described procedure). In animal that survived for thirty days, microsection discloses fibrous tissue infiltration of pancreas; two intact islets are shown, surrounded by normal-appearing acini. × 150.

11. Injection into gallbladder under pressure, of bile plus Staphlococcus flavus culture; cat (Archibald,[19] 1919).
12. Ligation of common bile duct distal to entrance point of pancreatic duct; goat (Mann and Giordano,[20] 1923).
13. Ligation of pancreatic duct plus administration of fatty meal; cat (Wangensteen and others,[21] 1931).
14. Injection of pancreatic duct with B. coli bullion culture and "infected" bile; dog (Kusunoki,[22] 1936).
15. Injection of mercury droplets into pancreatic artery; dog (Smyth,[24] 1940).
16. Ligation of pancreatic duct plus stimulation of vagus nerves; cat (Florey and others,[25] 1940).
17. Ligation of pancreatic duct plus administration of pilocarpine; dog (Coffey,[23] 1942).
18. Ligation of pancreatic duct plus administration of secretin, pilocarpine, acetylcholine and eserine; cat (Lium and Maddock,[26] 1948).
19. Ligation of pancreatic duct, impairment of vascular (blood) supply and administration of secretin; dog (Popper, Russell, and Necheles,[27] 1948).
20. Prolonged electrical stimulation of left splanchnic nerves; dog (Mallet-Guy,[28] 1949).
21. Repeated subcutaneous injection of mecholyl in a beeswax—mineral oil mixture; dog (Wener and others,[29] 1950).
22. Intraperitoneal injection of ethionine (α-amino-γ-ethylthiobutyric acid); rat (Farber and Popper,[30] 1950).
23. Injection of pancreatic duct with sodium taurocholate and commercial trypsin; dog (Ripstein and Thompson,[10] 1950).
24. Injection of pancreatic duct with autogenous bile (modified after Lewis and Wangensteen, 1950); dog (Persky and others,[31] 1951).
25. Ligation of pancreatic duct plus evaporated milk feedings; dog (Schaffarzick and others,[32] 1951).
26. Ligation of pancreatic duct and injection of saline solution under pressure; dog (Gibbs and Ivy,[33] 1951).
27. Intraperitoneal, subcutaneous and oral administration of ethionine; dog (de Almeida and Grossman,[34] 1952).
28. Crushing of pancreatic tissue; dog (Thistlewaite and Hill,[35] 1952).
29. Repeated traction on periductal (biliary and pancreatic) nylon sutures; dog (Waite,[11] 1952).
30. Injection of pancreatic duct with autogenous bile; dog (Hoffman and others,[36] 1953).
31. Pancreatic ischemia produced by temporary or permanently compromising the blood supply with ligature or clamp; rat (Block and others,[37] 1954).
32. Local pancreatic application of meningococcic toxin, followed by parenteral administration of the toxin (a Schwartzmann reaction); rabbit (Thal and Brackney,[38] 1954).
33. Pancreatic and bile duct interanastomosis plus administration of morphine (to produce spasm of sphincter of Oddi); dog (Powers and others,[39] 1955).
34. Ligation of bile duct, pancreatic resection, and retrograde pancreatijejunostomy on Roux-en-y principle; goat (Whitrock and others,[40] 1955).
35. Diversion of bile flow into pancreatic duct system; dog (Cross and others,[41] 1955).
36. Injection of bacteria into pancreatic duct; dog (Thal and others,[42] 1956).
37. Injection of pancreatic duct with dicetyl phosphate plus placement of "Ameroid" collar around pancreatic duct; dog (Floyd and Christophersen,[43] 1956).
38. Pancreaticocholedochostomy; anastomosis of main pancreatic duct to common bile duct; production of duct spasm by administration of morphine (Stein et al.,[44] 1956).
39. Isolation and closure of duodenal loop; gastroduodenostomy bypass, and division-ligation of common bile duct; dog (Pfeffer and others,[45] 1957).
40. Ligation of pancreatic duct and pancreatic vascular supply; dog, cat (Menguy and others,[46] 1957).
41. Injection of pancreatic duct with bile, trypsin, and/or decholin; dog (Anderson and others,[47] 1958).
42. Pancreatic (interstitial) injection with trypsin and trypsinogen; dog (Keith and others,[48] 1958).

NOTE 2.—Armour's Tryptar®: Purified crystalline trypsin derived from mammalian pancreas glands. Stable in dry form. When

placed with buffered phosphate solution, 75 per cent of proteolytic activity is dissipated in three hours. Effective range is *p*H 5 to 8.

NOTE 3.—Armour's buffered phosphate solution (Sorensen's), buffered at *p*H 7.1. Prepared by dissolving anhydrous dibasic sodium phosphate, 6.6 mg. per cubic centimeter, and anhydrous monobasic potassium phosphate, 2.7 mg. per cubic centimeter, in water.

NOTE 4.—Determination serum amylase (Krautman method): The Folin and Wu method of blood dextrose determination is modified by the use of a special deproteinizing solution; the normal-average serum amylase activity is 70 to 160 units per 100 cc. of blood serum (Am. J. Clin. Path. 3:9, 1939; Tech. Suppl.).

NOTE 5.—Blood sugar determination: The modified Folin and Wu method was used (*J. Biol. Chem.* 38:81, 1919, and 67:357, 1926).

NOTE 6.—The study is charted and summarized in the accompanying table.

Résumé of Experimental Study

	Number of Surgical procedures (dogs)	*Died*	*Survived*
Experimental pancreatitis (induced as described in text)	10	7	3
Experimental pancreatitis plus vagisection	10	9	1
Experimental pancreatitis plus vagisection 30 days prior to induction of disease	10	7	3
Experimental pancreatitis plus external pancreatic ductal drainage	10	8	2
Experimental pancreatitis plus pancreatic resection and intravenous infusions (12 hours after pancreatitis was induced)	7	7	0

SUMMARY

A method of producing acute hemorrhagic pancreatitis in the dog is described; no originality is claimed as to its mode of application. The method has merit in that it embodies the predominant concept of the pathogenesis of acute hemorrhagic pancreatitis; it consistently produces the same lesions each time it is applied, and it has inherent simplicity. This simplicity has led the authors to the opinion that it could serve as an experimental tool for investigators, as well as a teaching exercise for surgical resident trainees. It is suggested that the multiple and varied experimental procedures previously reported may have had a tendency to confuse and delay a long-needed concrete effort to find a definitive therapeutic approach to this disease in man. The problem of reducing the present significant morbidity and mortality rates should center around efforts to prevent the noxious products of pancreatic tissue necrosis from being absorbed by the peritoneal serosa and thus gaining the systemic vascular circulation.

ZUSAMMENFASSUNG

Der Verfasser beschreibt ein Verfahren zur Erzeugung einer akuten hämorrhagischen Bauchspeicheldrüsenentzündung beim Hunde, ohne Anspruch auf Originalität der Anwendung der Methode zu erheben. Das Verfahren hat das Verdienst, die vorherrschende Auffssung von der Pathogenese der akuten hämorrhagischen Bauchspeicheldrüsenentzündung zu verköpern, bei jedesmaliger Anwendung stets die gleichen Veränderungen hervorzurufen und sich durch eine ihm eigene Einfachheit auszuzeichnen. Wegen dieser Einfachheit wird die Methode sowohl als Werkzeug experimenteller Forschung als auch als Lehrübung für chirurgische Assistenten für geeignet gehalten. Es wird angedeutet, dass die vielfachen verschiedenen früher veröffentlichten Untersuchungsverfahren vielleicht dazu beigetragen haben, einen lange begehrten greifbaren Ansatz zur Entdeckung eines entschiedenen Behandlungsplanes dieser Erkrankung beim

Menschen zu stören und zu verzögern. Die Bemühungen um eine Herabsetzung der gegenwärtigen erheblichen Krankheitsund Sterblichkeitsziffern sollten sich darauf konzentrieren, die Absorbierung der schädlichen Erzeugnisse pankreatischer Gewebsnekrose durch die Bauchfellserosa zu verhindern und ihren Eintritt in die allgemeine Blutzirkulation zu vermeiden.

RÉSUMÉ

Une méthode permettant de produire une pancréatite hémorragique aiguë chez le chien est décrite; l'auteur n'a aucune prétention à l'originalité quant à son mode d'application. Le mérite de cette méthode est de faire ressortir la cause principale de la pathogénèse de la pancréatite hémorragique aiguë. Elle produit uniformément les mêmes lésions chaque fois qu'elle est appliquée et elle est d'une grande simplicité, ce qui incite les auteurs à penser qu'elle peut servir d'instrument de travail pour les expérimentateurs aussi bien que de moyen d'enseignement pour les internes de chirurgie. Le auteurs pensent que les techniques expérimentales multiples et variées rapportées antérieurement peuvent engendrer la confusion et retarder un effort de recherche depuis longtemps nécessaire en vue de trouver une voie d'approche bien définie de cette affection chez l'homme. Le problème de la réduction du taux élevé de morbidité et de mortalité devrait être centré autour des efforts tendant à éviter que les produits toxiques provenant de la nécrose du tissu pancréatique soient absorbés par la séreuse péritonéale et entraînés dans la circulation vasculaire.

RESUMEN

Se describe en este trabajo un método de provocar en un perro una pancreatitis aguda hemorrágica. No se trata de un método original. El mérito del método consiste en que concierne al concepto dominante de la patología de la pancreatitis hemorrágica aguda; el métod produce sistemáticamente las mismas lesiones cada vez que se emplea y tiene una gran simplicidad. Tal simplicidad lleva a los autores a la conclusión de que puede ser empleado como norma experimental para los investigadores así como al ejercicio de la enseñanza para futuros cirujanos. También opina el autor que las diversos y múltiples procedimientos experimentales conocidos con anterioridad, tendían a la confusión y a retrasar un esfuerzo aunado por encontrar un método terapéutico definitivo para esta enfermedad. El problema debe centrarse en reducir las cifras actuales de morbilidad y mortalidad evitando que los productos tóxicos del tejido pancreático necrosado absorben la serosa peritoneal y entren en la circulación vascular sistémica.

SUMARIO

Descreve um metodo para produzir pancreatite aguda hemorragica em cães. Esse método tem o mérito de enfeicar o conceito predominante da patogenia e consiste em produzir a mesma lesão cada vez que é aplicado com grande facilidade. Essa simplicidade pensa o A. poderá servir de meio para investigação bem como para motivo detreinamento de residentes. Acha que os metodos anteriormente descrito são multiplos e complexos levando a confusão e perda de tempo. O problema de reduzir a atual taxa de morbidade e mortalidade deve convergir para prevenir a produção de produtos toxicos da necrose pancreatica que são levados á corrente circulatoria pela absorção.

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In order to comprehend the role played by Renaissance art in the development of medicine, we should remember that medical science and art have been closely related for long periods in history. In ancient civilizations, medicine, art, and religion formed an indissoluble trinity. The priest of primitive cultures was also the medicine man who, besides acting as an intermediary between the gods and the people, used his influence with the deities to cure the sick. He was also the first patron of the arts, since he had to present the deities to his people in the most artistic form, which forced him to seek artists who could fashion the best images of the gods. The priest, therefore, was not only a healer but also a Maecenas.

In the Middle Ages, the trinity disintegrated and the medicine man and the artist became specialized figures, although art continued subserving religion. But in the Renaissance, art and medicine were again united. The need for representing the parts of the body with exactness forced the artist and the physician to become one and the same. But until dissections were authorized, the artist was forced to use diagrams of animals or to make imaginary drawings of the human body.

—*Marti—Ibañez*

Surgical Anatomic Aspects of the Anorectal Sphincter Mechanism and its Clinical Significance

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The surgical anatomic aspects of the anorectal sphincter mechanism are reviewed on the basis of histologic examination of the anorectal region. Emphasis is laid on the clinical importance of a previously undescribed interstitial space, the anal intermuscular interstice, which may be regarded as the gateway to the perianal region and the ischioanal fossa. The fact that each sphincter muscle has its associated longitudinal muscle provides an anatomic explanation of reciprocal innervation and the development of anal incontinence after fistulectomy, an iatrogenic complication that can be prevented by preservation of the sphincter mechanism as a functional anatomic unit.

THE concept of the anorectal sphincter mechanism as a composite structure, consisting of several muscles, was proposed by Milligan and Morgan.[1] This structure is usually described as consisting of four muscles, namely, the external sphincter, the internal sphincter, the conjoined longitudinal muscle and the puborectalis division of the levator ani. As will be seen, four additional muscles should be considered. Along with the muscularis submucosae ani, one should include the attachments of the anorectal sphincter mechanism posteriorly to the rectococcygeus muscle and anteriorly to the muscles at the central point of the perineum, e.g., the deep transverse perineal muscle, and the rectourethralis in the male or the rectovaginalis muscle in the female.

In spite of many important contributions to current knowledge of the surgical anatomic aspects of the anal canal, the literature is characterized by conflicting opinions, ambiguous terms and erroneous illustrations. The present study, based on the histologic examination of postmortem and surgical specimens of the anorectal region in 6 adults, 1 child and 8 newborn infants, was undertaken to gain a clearer understanding of the anatomic relations and to correlate these observations with certain clinical aspects of proctology.

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Anatomic Relations.—The anatomic relations of the muscles constituting the anorectal sphincter mechanism can be demonstrated by histologic examination of this region in the newborn (Fig. 1). The homogeneous character of this composite structure appears to be related to the distribution of the fibers of the conjoined longitudinal muscle, which acts as a vinculum, sending its interlacing fibers into both the circular muscle coat of the rectum and the external sphincter, giving rise to

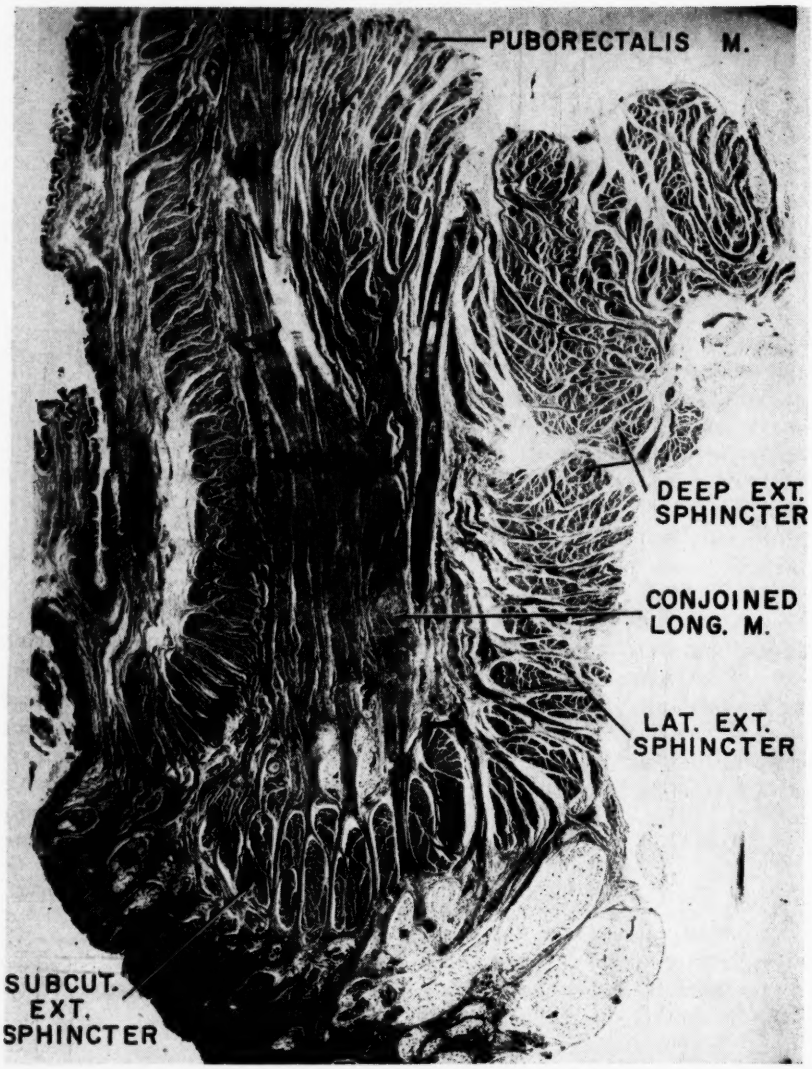

Fig. 1.—Sagittal section of entire anorectal sphincter mechanism (newborn). Note manner in which deep, lateral and subcutaneous parts of external sphincter become fused with hindgut by septums of conjoined longitudinal muscle which penetrate into ischioanal fossa and are inserted into perianal skin.

a sphincter mechanism composed of both voluntary and involuntary muscles.

The Muscularis Submucosae Ani and the Internal Sphincter: In 1923, in his system of anatomy, Tandler[2] described a group of longitudinal muscle fibers that penetrate through the circular muscle coat to give rise to the substratum of the columns of Morgagni (Fig. 2). This can be confirmed by histologic examination of this region through the frontal plane in the adult (Fig. 3). Fine and Lawes[3] also

demonstrated well-developed involuntary muscle fibers in the submucosa of the anal canal, to which they applied the term "muscularis submucosae ani." Wilde[4] revealed that these fibers are derived from two sources. The present study confirms these observations (Fig. 4). The more superficial fibers are continuous with the muscularis mucosae of the rectum, while the deeper fibers are derived from the conjoined longitudinal muscle and penetrate between the bundles of the circular muscle to fuse with the superficial stratum, as they are traced distally in the anorectal submucosa.

The internal sphincter should not be construed as merely the thickened termination of the circular muscle coat. In the adult, according to Wilde,[4] the internal sphincter measures 30 to 35 mm. in length and 5 to 8 mm. in breadth. The upper level of the internal sphincter is marked by the penetration of the fibers of the muscularis submucosae ani, which corresponds roughly with the levator-rectal juncture. It is of interest that the fibers of the thickened termination of the internal sphincter are separated by the downward projection of the fibroelestic septums of the muscularis submucosae ani (Fig. 5). These penetrating fibers pass downward through the terminal internal sphincter to emerge between the apposed ends of the internal and external sphincter muscles, where they give rise to the so-called anal intermuscular septum and fuse at the mucocutaneous juncture with the submucosal fibers passing medial to the internal sphincter. It should be noted that the attenuated epithelium at the point of insertion into the mucocutaneous juncture may explain the "white line of Hilton"[5] and represents one of the sites of fusion of the hindgut with the cloacal membrane.

The Anal Glands: Between each two adjacent columns of Morgagni lies the anal crypt (Fig. 3). These pocketlike depressions can be seen to deepen into the distinct open and closed pouches (Fig. 6) first described in 1880 by Hermann and Desfosses,[6] who referred to them as anal glands in the anorectal submucosa with

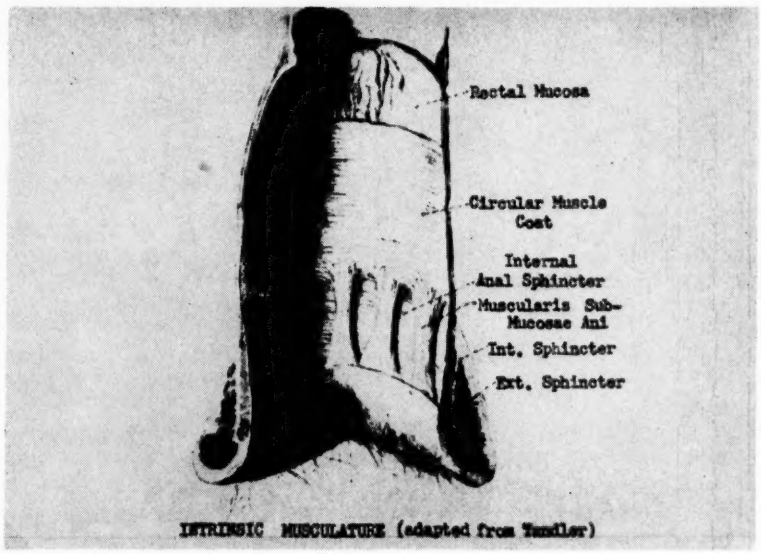

Fig. 2.—Intrinsic musculature of anorectal region (adapted from Tandler).

Fig. 3.—Frontal section (adult), showing how columns of Morgagni are formed by longitudinal fasciculi of muscularis submucosae ani breaking through circular muscle. Note anal crypt (*A*) between columns.

occasional extension into the internal sphincter and emphasized their surgical importance in the pathogenesis of anorectal fistula. In recent years, Tucker and Hellwig[7] and others,[8] have reinvestigated these structures, designating them, the "anal glands and ducts." The present investigation reveals that these saccular structures are intimately related to the fibroelastic fibers in the anorectal stroma

(Fig. 7) and appear to result from the downward prolongation of the cloacal membrane along with the mucosa of the lower rectum associated with the descent of the longitudinal fasciculi of the muscularis submucosae ani from the hindgut. Coronal sections through the level of the anal crypts reveal that these branched, tubular glands are lined by columnar epithelium in their depths with an abrupt change to transitional epithelium as these structures approach the surface (Fig. 8). The presence of the two types of cells in these glands may explain the development of adenocarcinoma in the anal crypts and the various anorectal carcinomas of extramucosal origin, as described by Zimberg and Kay.[9]

The External Sphincter and the Conjoined Longitudinal Muscle: The external sphincter represents, on either side, the downward prolongation of the puborectalis muscle. These muscles are fusiform in outline, flattened, and obliquely placed about the anus and anal canal. Anteriorly, the two muscles join in the region of the median raphe, where their deep fibers become fused with the posterior aspect of the deep transverse perineal muscle at the central point of the perineum. This is well illustrated in the male (Fig. 9), where the rectourethral muscle can be seen as the downward and forward continuation of the conjoined longitudinal muscle, blending with the other muscles at the central point of the perineum, as it passes to its insertion in the urethra. As was pointed out by Gorsch,[10] this anterior point

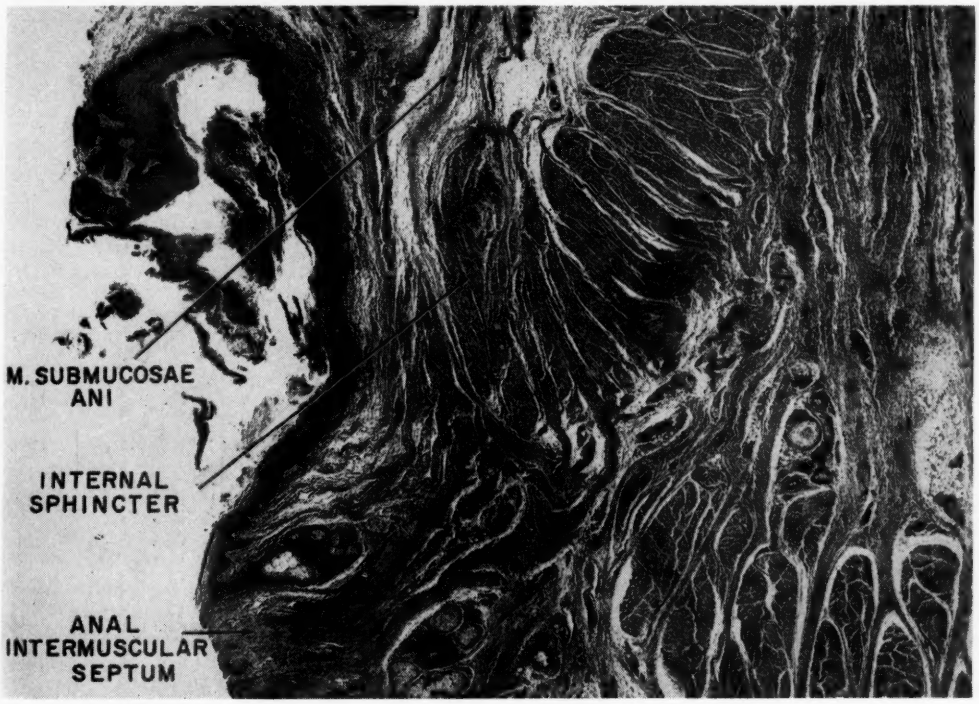

Fig. 5.—Anal intermuscular septum. Note how fibers of muscularis submucosae ani penetrate terminal internal sphincter and pass between apposed edges of internal and external sphincters to join with medial flaps at mucocutaneous juncture.

of juncture is the fulcrum around which the anorectal and genitourinary musculature coordinate their functional activities.

It would appear that we are plagued with the common misconception that the fibers of the external sphincter muscle from each side converge posterior to the anal canal, permitting the formation of a potential space. The fibers of these muscles, however, not only converge but fuse, at their insertion into the anococcygeal raphe, from the anal canal to the tip of the

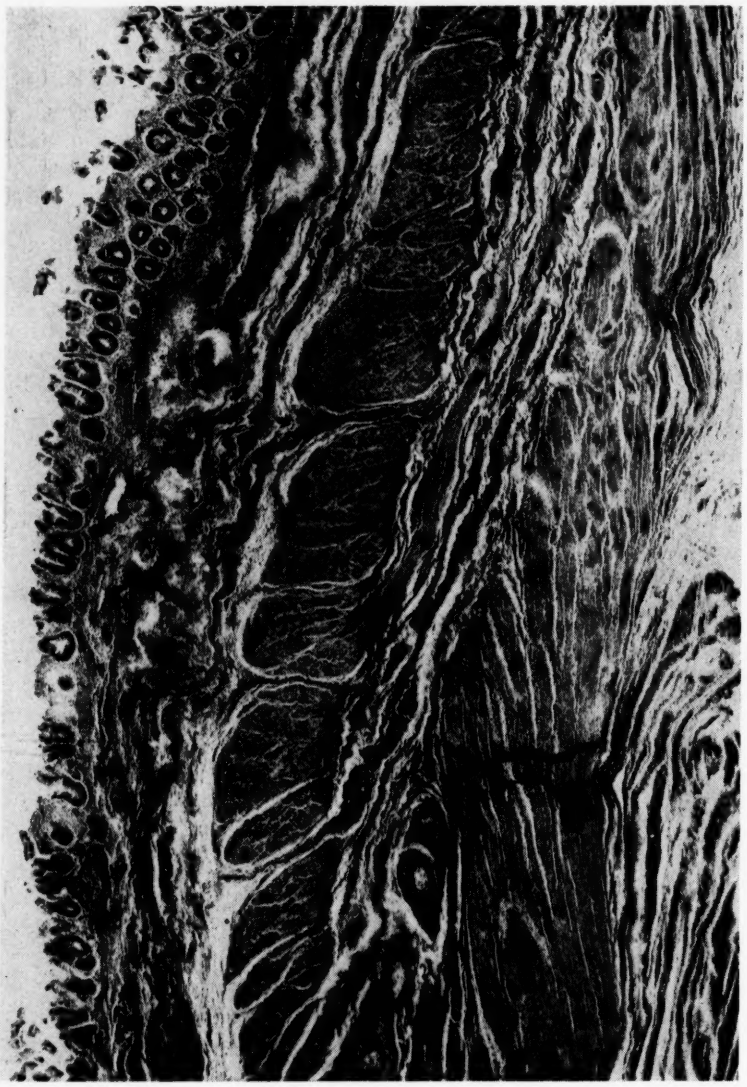

Fig. 4.—Sagittal section (newborn), revealing derivation of muscularis submucosae ani from muscularis mucosae of rectum and conjoined longitudinal muscle.

Fig. 6.—Sagittal section (newborn), showing anal glands.

coccyx[11] (Fig. 10). This fusion is noted in the subcutaneous part of the external sphincter by the decussation of the fibers in the anterior and posterior midlines. Not only are the fibers of the conjoined longitudinal muscle more compact posteriorly in the median plane (Fig. 11), but at a higher level they blend with voluntary muscle fibers from the anterior surface of the coccyx to form the rectococcygeus muscle. The compactness of the fused fibers along this posterior attachment of the sphincter mechanism, makes it difficult to confirm the presence of the posterior triangular space described by Brick.[12]

As is demonstrated in Figure 1, the ex-

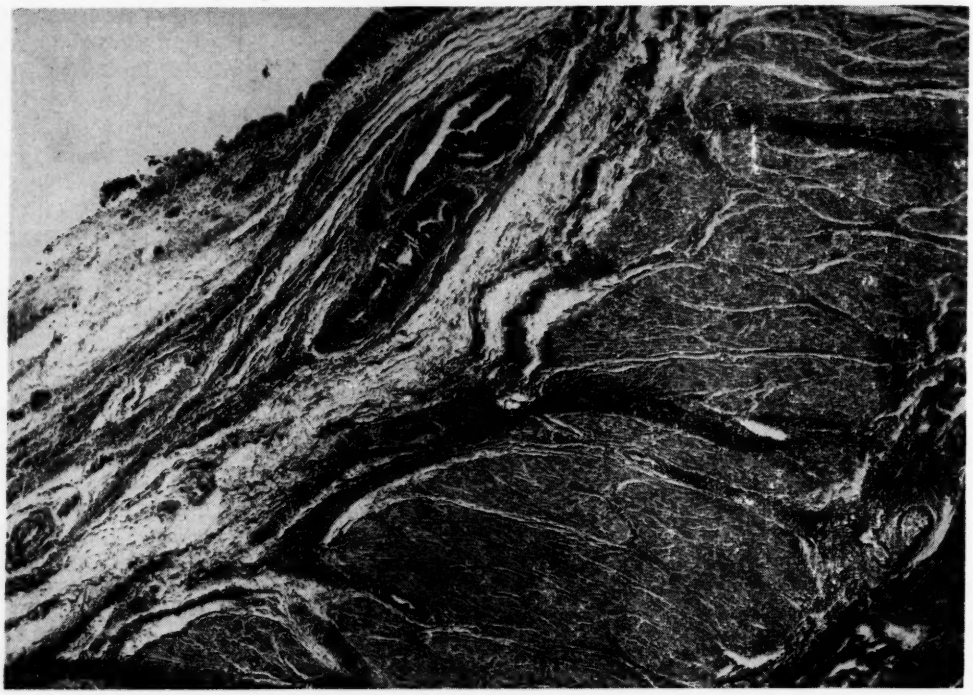

Fig. 7.—Sagittal section (newborn), revealing anal glands embedded among fibers of muscularis submucosae ani.

ternal sphincter is not divided into three components, which confirms the observations of Blaisdell[13] and more recently those of Goligher, Leacock and Brossy.[14] On either side, this muscle descends to occupy a position below and lateral to the internal sphincter. As it descends, it becomes fused with the hindgut by the septate terminations of the conjoined longitudinal muscle, which penetrate the external sphincter and divide it into numerous bundles. These septate fibers penetrate laterally beyond the external sphincter, passing into the ischioanal fossa, and insert into the perianal skin below the subcutaneous portion of the external sphincter. It is this insertion into the perianal skin which is responsible for the characteristic puckering of the anus. As was pointed out by Wilde,[4] there is no evidence to confirm the existence of a separate corrugator cutis ani muscle.

It is to be noted that each anal sphincter has its respective associated longitudinal muscle, the muscularis submucosae ani being distributed to the internal sphincter, while the conjoined longitudinal muscle passes to the external sphincter. The sphincters of the bowel, e.g., the ileocecal and the internal and external anal sphincters, according to Best and Taylor,[15] exemplify the principal of reciprocal innervation. A similar interlacing of the longitudinal fibers with the circular muscle at the ileocecal valve has been demonstrated by Toldt.[16] There appears to be a definite relation between this anatomic structural pattern and sphincter activity, providing an anatomic explanation for reciprocal innervation. Thus, stimulation of

the parasympathetics causes evacuation as the result of strong peristaltic contraction of the bowel, accompanied by contraction and shortening of its longitudinal fibers, which elevate and open the sphincters, giving rise to an apparent relaxation.

The Anal Intermuscular Interstice: It will be noted that at the level of the deep fibers of the external sphincter the longitudinal muscle of the rectum becomes fused with the fascia of the puborectalis muscle (Fig. 1). This conjoined longitudinal muscle becomes broader and increasingly fibroelastic as it descends. Just before it approaches the subcutaneous portion of the external sphincter muscle, the conjoined longitudinal muscle coat can be seen to end, terminating in multiple filamentous, fibroelastic septums which traverse a definite interstitial space before they proceed to divide the external sphincter into numerous bundles (Figs. 12 and 13). This previously undescribed *anal intermuscular interstice* almost completely encircles the anorectal region and has the following boundaries: the termination of the conjoined longitudinal muscle coat superiorly, the external sphincter muscle laterally and inferiorly and the internal sphincter medially. It is contiguous with the fibroelastic fibers of the muscularis submucosae ani, as they penetrate the internal sphincter muscle to form the anal intermuscular septum. In the region of the anococcygeal raphe and the median raphe, however, this tissue interval becomes obscured by the compactness of the septums of the conjoined longitudinal muscle. Coursing through this interstitial space, in addition to the septate fibers, are nerves, blood vessels and lymphatics surrounded by adipose tissue.

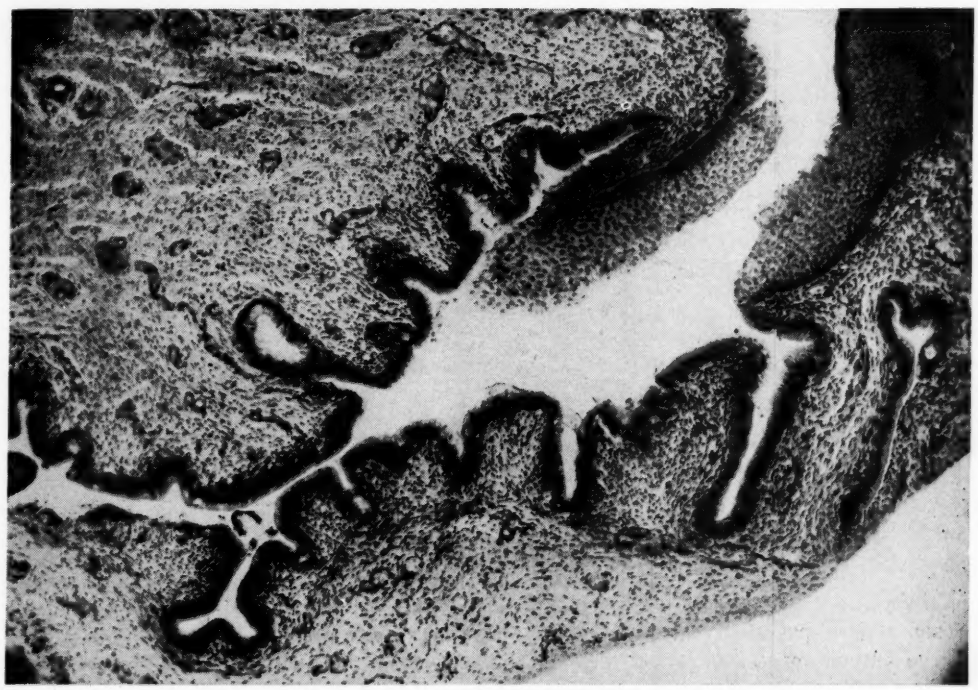

Fig. 8.—Coronal section through anal crypts (newborn), showing columnar cells in depth of anal gland, with abrupt change to transitional epithelium near surface.

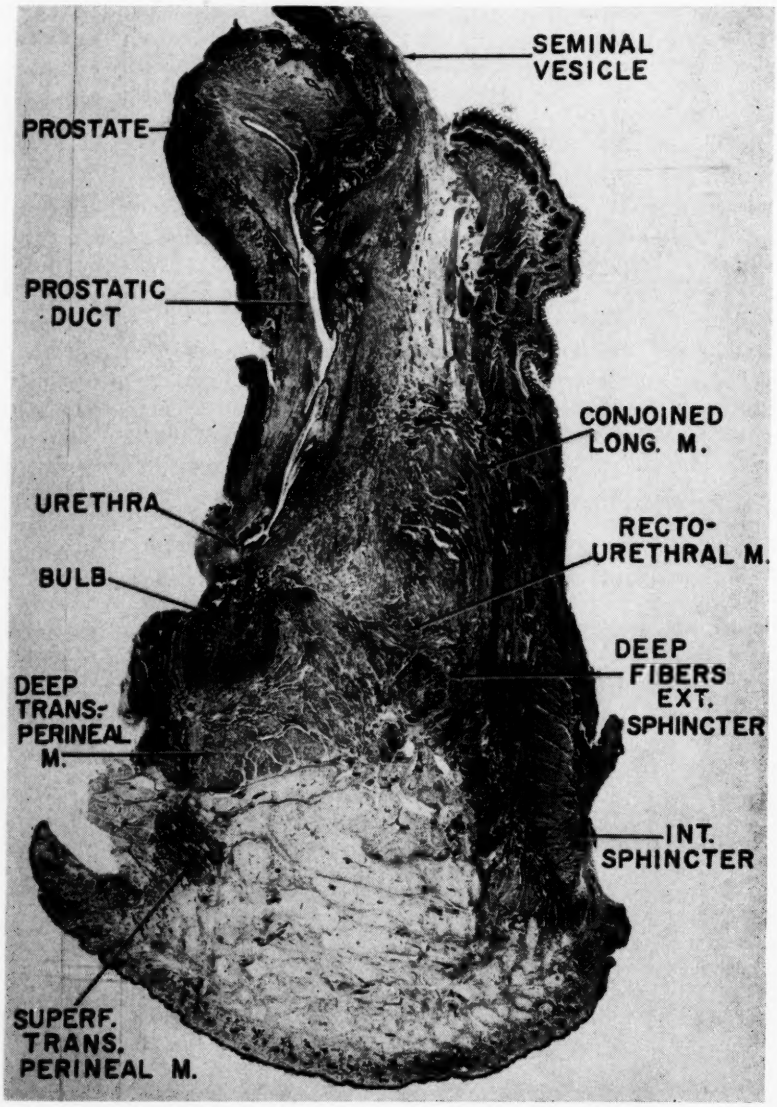

Fig. 9.—Sagittal section through median raphe in male (newborn), showing anterior relations. Note also compactness of conjoined longitudinal muscle and fibroelastic septums passing to skin.

Clinical Considerations.—The anal canal presents, perhaps, one of the best examples of the important relation between anatomic structure and the clinical manifestations of disease. The clinical significance of the anorectal sphincter mechanism appears to be related to the distribution of its longitudinal fibers, which plays an important role in anorectal suppurative disease, in the spread of anorectal malignancy

and in the development of hemorrhoidal varicosities, as well as of postoperative anal incontinence.

Anorectal Suppurative Disease: Over the years, the proctologic surgeon has sought to explain the course of anorectal fistulas. Much emphasis has been placed on the role of the anal glands in this process. The present study reveals how an inflammatory lesion, arising in an infected anal crypt (Fig. 14), burrows through the fibers of the internal sphincter along the fibroelastic septums of the conjoined longitudinal muscle to gain access to the anal intermuscular interstice. The suppurative process may find egress from this interstitial space into either the perianal region or the ischioanal fossa (Fig. 12). While in some cases the inflammatory process may follow the septums that divide the external sphincter muscle, the spread of infection is usually determined by the distribution of the lymphatics and veins. This opinion finds support when one considers the symmetric course of horseshoe fistulas. In the perianal region the septums would appear to confine the suppurative process, giving rise to a "closed space infection." As demonstrated, the intimate relation between the anal glands and the submucosal septate fibers would seem further to confirm the role of these structures in the spread of anorectal infections, especially those involving the more superficial, terminal fibers of the internal sphincter muscle.

The upward spread of an inflammatory process can now be accounted for, as it burrows along the septums in the submucosal tissues, or in the interval between

Fig. 10.—Coronal section through level of anal crypts, showing how fibers of external sphincter muscle from each side converge and become fused in posterior midline.

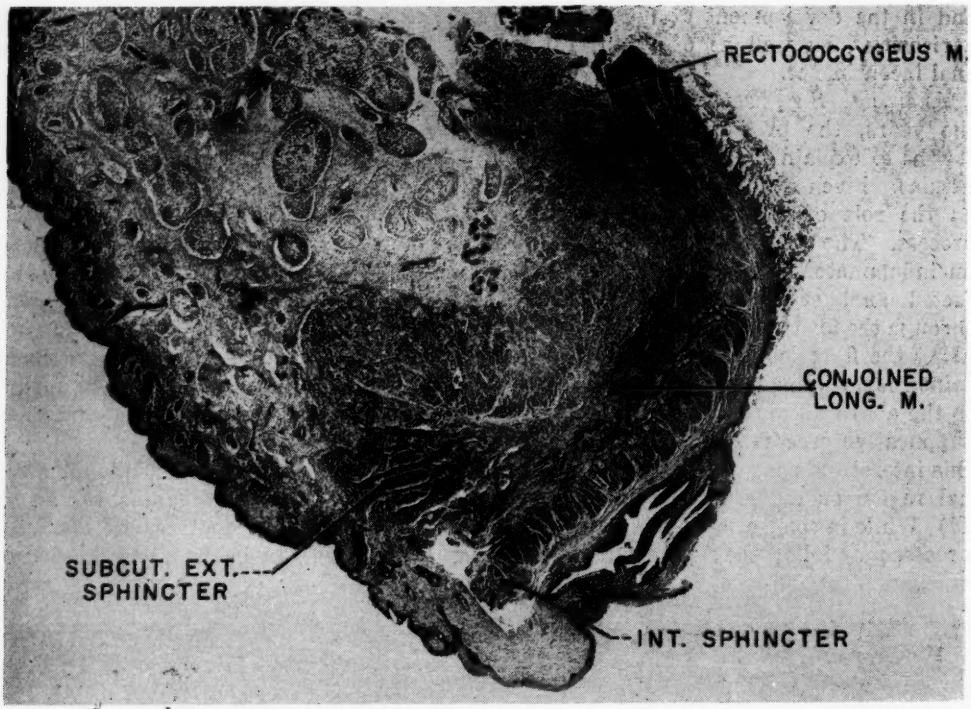

Fig. 11.—Sagittal section through anococcygeal raphe, showing compactness of conjoined longitudinal muscle, which, at higher level, blends with voluntary muscle fibers to form rectococcygeus muscle.

the circular and the conjoined longitudinal muscle, giving rise to either a submucous or an intermuscular anorectal abscess. This study also reveals how an infection may extend along the fibroelastic septums of the muscularis submucosae ani medial to the internal sphincter (Fig. 5), with little or no involvement of the sphincter mechanism, to exteriorize in the region of the mucocutaneous juncture. In an experience covering a number of years, I do not recall having noted an external sinus of an anorectal fistula in the anal fold or in the median raphe, except those sinuses at the anal verge associated with superficial subcutaneous fistulas. This may be explained by the compactness of the fibers of the conjoined longitudinal muscle, with obliteration of the anal intermuscular interstice at these points of fusion anterior and posterior to the anus. The tracts of deeper fistulas are generaly directed to the right or left of the anterior or posterior midlines, determined by the course of the lymphatics and veins.

The Spread of Anorectal Malignant Disease: Since the anal intermuscular interstice may be regarded as the "gateway" to the ischioanal fossa and the perianal region, its possible role in the spread of anorectal carcinoma was considered. Grinnell[17] has shown that involvement of the sphincter muscles and the perianal tissues occurs frequently in association with squamous cell carcinoma of the anus. Metastases in such cases appear to be most frequently confined to the downward and lateral zones of spread described by Miles[18] and to the inguinal nodes. Figure 15 illustrates the manner in which an

adenocarcinoma of the anal crypt infiltrates laterally through the fibers of the internal sphincter muscle to spread intramurally, upward or downward, along the fibers of the conjoined longitudinal muscle. The downward spread of the carcinoma into the upper reaches of the anal intermuscular interstice can be observed, providing access to the inferior group of lymphatics which drain the anal canal and empty into the inguinal lymphatics. This observation suggests that, in determining the prognosis and the indication for inguinal node dissection, the entire anorectal sphincter mechanism should be examined histologically through the sagittal plane. Inguinal node involement may be anticipated if the carcinoma has infiltrated into the anal intermuscular interstice. The compactness of the fibers of the conjoined longitudinal muscle at the anterior and posterior points of fusion, with obliteration of the anal intermuscular interstice, may explain the relatively infrequent and late involvement of the prostrate, vagina, bladder and sacrum.

Hemorrhoidal Varicosities: In a previous communication[19] I postulated that, because of the rich anastomosis in the anal canal between the superior hemorrhoidal veins and the middle and inferior hemorrhoidal veins, one would assume that adequate drainage of this region should take place. It becomes apparent that hemorrhoidal varicosities are not the result of incompetent check valves in the superior hemorrhoidal veins alone. The implication is clear that there must be, at least, partial blockage of the tributaries of the middle and inferior hemorrhoidal veins.

Fig. 12.—Anal intermuscular interstice. Note termination of conjoined longitudinal muscle and nerve and blood vessels in this tissue interval.

Fig. 13.—Frontal section (adult), showing anal intermuscular interstice. Note terminal fibers of conjoined longitudinal muscle above, with fibers of external sphincter below.

Internal hemorrhoids can be shown to be intimately related to the fibers of the muscularis submucosae ani that form the substratum of this type of lesion (Fig. 16). Either as the result of functional hypertrophy associated with the act of defecation and/or fibrosis related to chronic inflammation, thickening of these fibroelastic submucosal fibers occurs. This hypertrophy and/or fibrosis of the muscularis submucosae ani could provide an effective barrier to the tributaries of the

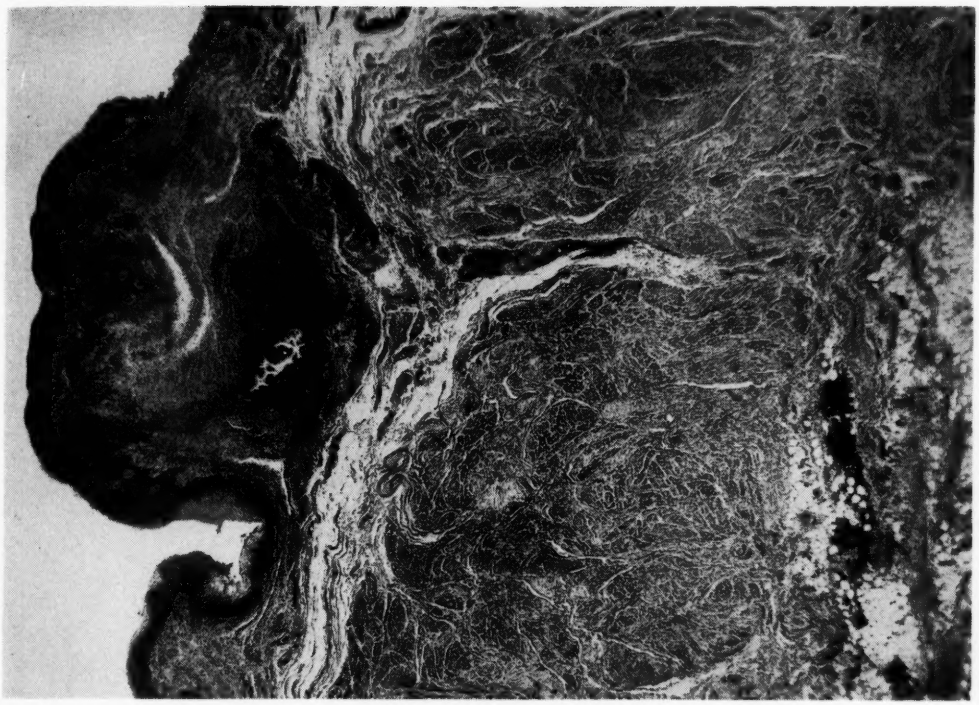

Fig. 14.—Infected anal crypt. Note manner in which inflammatory process burrows through fibers of internal sphincter to gain access to anal intermuscular interstice.

veins of the caval system, interfering with the venous drainage of this region. It is this muscle, aided by the internal sphincter and the conjoined longitudinal muscle, which is principally responsible for the spontaneous return of a protruded hemorrhoid.

Postoperative Anal Incontinence: This discussion is limited to a consideration of anal incontinence incident to the injudicious division of the anorectal sphincter mechanism during the performance of fistulectomy, giving rise to either total or partial fecal incontinence. Fortunately, total incontinence is much less frequent than partial incontinence, which is associated with limited control of the seepage of liquid feces and the passage of flatus. These iatrogenic tragedies are avoidable and may in part be explained by blind acceptance of the dictum of Milligan and Morgan[1] that, if any part of the anorectal ring lies above the probe inserted into the tract of the fistula, section of the whole muscle lying below the probe may be carried out. With this opinion, however, I take issue, since it implies impunity in severing the important distal fibers of the anorectal sphincter mechanism. When one considers the significance of the anal intermuscular interstice in the morbid anatomic changes associated with anorectal fistulas, it becomes obvious that it is the lower fibers of this structure that are involved in the suppurative process. From a functional standpoint, the architecture of this composite structure is such that the juncture of the apposed edges of the internal and external anal sphincters at the anal intermuscular septum is of prime

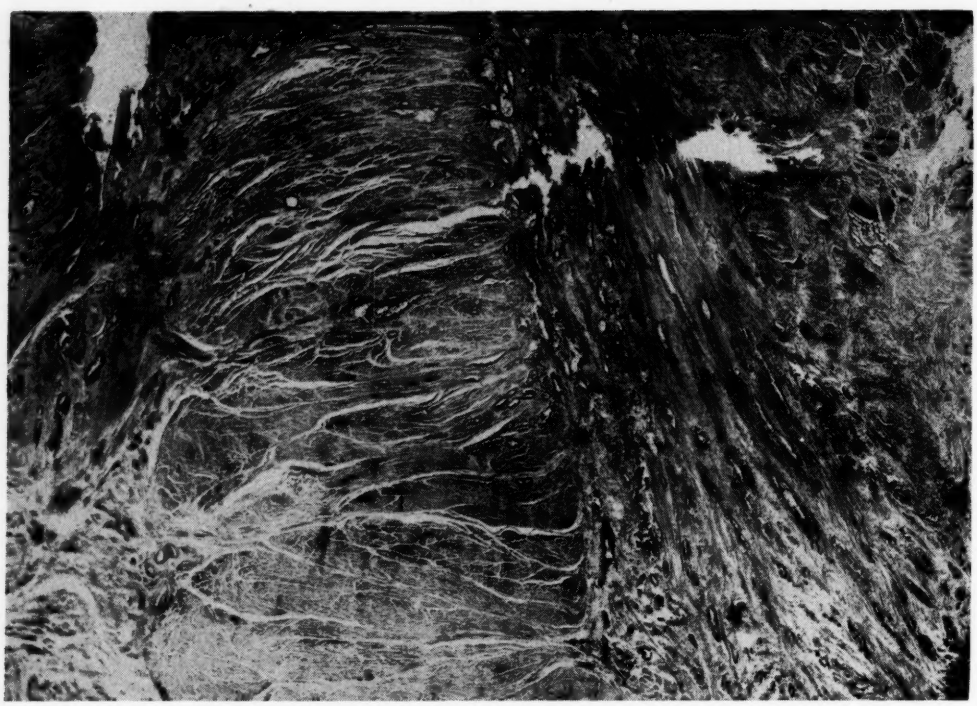

Fig. 15.—Adenocarcinoma of anus, revealing manner of spread through sphincter mechanism. Note infiltration into anal intermuscular interstice (*lower right*).

importance in the reflex and voluntary control of the passage of flatus and feces (Fig. 12).

While the separation of these fibers and the development of the broad scar has been attributed by some to injudicious packing of the fistulectomy wound, postoperative anal incontinence appears to be the result of separation of the divided fibers of both the internal and external sphincters by the tonus and upward pull of their respective longitudinal muscles. The tonus of the conjoined longitudinal muscle is attested by persistent corrugation of the perianal skin, while that of the muscularis submucosae ani may be observed by the separation of the fibers of the internal sphincter during the performance of an internal sphincterotomy. Thus, the deeper the incision through the lower fibers of the anorectal sphincter mechanism, the greater the separation and the broader the scar.

Admittedly, the results following the various plastic procedures for the repair of anal incontinence are not brilliant. The proctologic surgeon should, therefore, be more concerned with the prevention of this avoidable complication. Injury to the anorectal sphincter mechanism can be minimized by the use of the "seton," a time-honored method dating back to Hippocrates.[20] The two-stage fistulectomy has been used and redescribed by many authors and is indicated in any case in which the fistulous tract is so situated that it is necessary to divide the lateral (superficial) and the subcutaneous fibers of the external sphincter muscle along with the internal sphincter. In more superficially

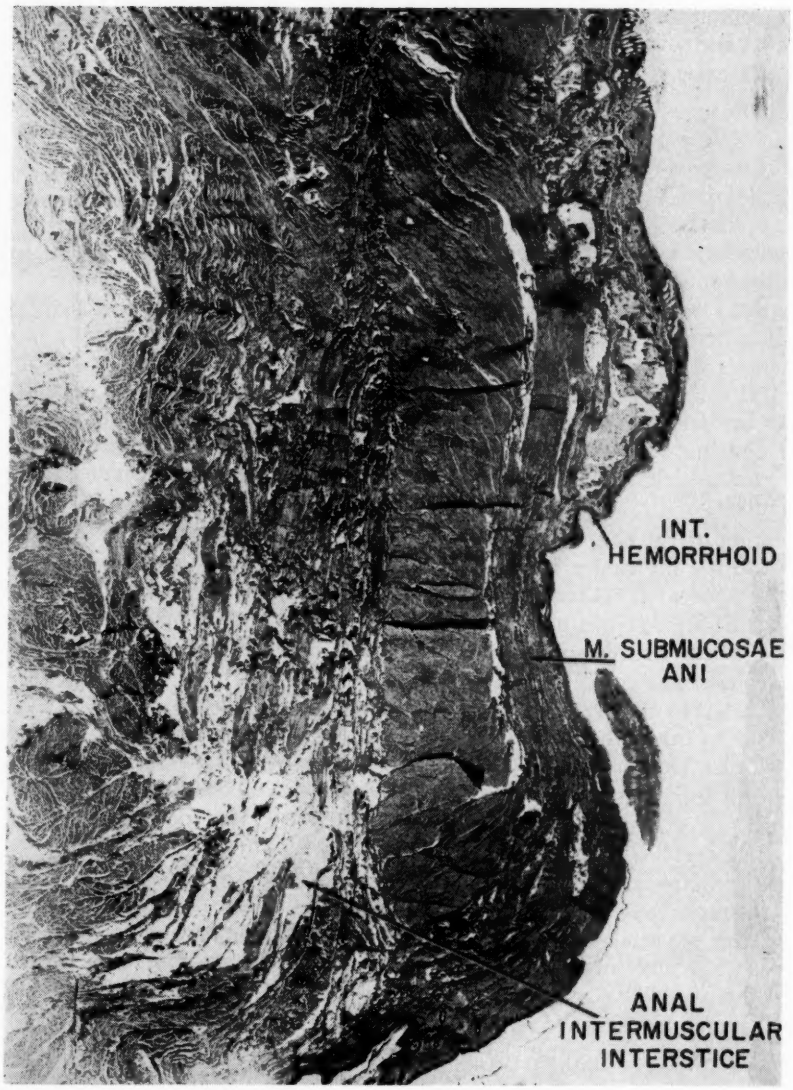

Fig. 16.—Sagittal section (adult), showing relation of internal hemorrhoid to underlying muscularis submucosae ani. Note also attachment of muscularis submucosae ani to terminal internal sphincter.

placed tracts the practice of carrying the fistulectomy through the sphincter muscles would appear unwise, since many of the muscle fibers are needlessly sacrificed during the procedure. When the dissection approaches the sphincter mechanism in such cases, the fistulectomy should terminate in a fistulotomy. I shall not enter into a discussion of the various alleged locations where the sphincter mechanism may be divided with the smallest loss of control, except to emphasize the fact that

this is a composite anatomic and functional unit, and every effort should be made to preserve it as nearly intact as possible.

SUMMARY

The surgical anatomic character of the anorectal sphincter mechanism has been reinvestigated and is redescribed on the basis of histologic examination of the anorectal region.

It has been pointed out that, in addition to the muscles usually mentioned as constituting this composite structure, it is important to include the muscularis submucosae ani and the muscles at the anterior and posterior attachments of the sphincter mechanism.

The relation of the conjoined longitudinal muscle to the internal phincter, to the formation of the anal intermuscular septum and to the anal glands has been demonstrated. These fibroelastic submucosal fibers have been shown to form the substratum of internal hemorrhoids, and their probable role in the pathogenesis of these varicosities has been suggested.

The clinical importance of a previously undescribed interstitial space, the anal intermuscular interstice, along with the septum of the conjoined longitudinal muscle, coursing through the internal sphincter, is emphasized, especially with reference to the spread of anorectal suppuration and malignant disease. The anal intermuscular interstice may be regarded as the gateway to the perianal region and the ischioanal fossa.

The architecture of the anorectal sphincter mechanism is such that each sphincter muscle has its associated respective longitudinal muscle, providing an anatomic explanation for reciprocal innervation and the development of postoperative anal incontinence incident to fistulectomy operations. Preservation of the sphincter mechanism as a functional anatomic unit in fistulectomies has been stressed in order to prevent this avoidable iatrogenic postoperative sequela.

Author's Note: I should like to acknowledge the cooperation of Dr. Medrick Barbot of the department of pathology of Provident Hospital and Dr. Nancy Warner of the department of surgical pathology, University of Chicago Clinics, in providing the material used in this study. I am also indebted to Mrs. Maxine Mitchell and Mrs. Anne B. White for the histologic preparations.

ZUSAMMENFASSUNG

Die chirurgisch-anatomischen Eigenheiten des anorektalen Schliessmuskelmechanismus werden auf Grund histologischer Studien der Anorektalgegend einer neuen Untersuchung unterzogen.

Es wird darauf hingewiesen, dass es wichtig ist, ausser den Muskeln, die gewöhnlich als Elemente der Zusammensetzung dieses Gebildes erwähnt werden, auch die Muscularis submucosae ani und die Muskeln des vorderen und hinteren Ansatzes des Schliessmuskelmechanismus zu berücksichtigen.

Die Beziehungen der Muscularis submucosae ani zum inneren Schliessmuskel, zur Bildung des intermuskulären Septums des Afters und zu den Analdrüsen werden dargestellt.

Es hat sich gezeigt, dass diese submukösen fibroelastischen Fasern das Substrat innerer Hämorrhoiden bilden. Es wird angedeutet, welche Rolle sie wahrscheinlich in der Entstehung dieser Venenerweiterung spielen.

Die klinische Bedeutung eines bisher nicht beschriebenen interstitiellen Raumes, des intermuskulären Interstitiums, und der durch den inneren Schliessmuskel verlaufenden Septen der Muscularis submucosae ani wird hervorgehoben unter besonderer Berücksichtigung des Ausbreitungsweges von Eiterungen und bösartigen Erkrankungen der Anorektalgegend. Das anale intermuskuläre Interstitium kann als "Eingangspforte" zur Perianal-

gegend und zur Fossa ischioanalis angesehen werden.

Der anorektale Schliessapparat ist so aufgebaut, dass zu jedem Schliessmuskel ein entsprechender Längsmuskel gehört, womit eine anatomische Erklärung für die wechselseitige Innervierung und für die Entwicklung der nach Fisteloperationen vorkommenden Schlussunfähigkeit gegeben ist. Die Wichtigkeit der Erhaltung des Schliessmuskelmechanismus als funktionelle anatomische Einheit bei Fistelresektionen wird besonders hervorgehoben, wenn man das Auftreten solcher unnötigen postoperativen dem ärztlichen Eingriff zuzuschreibenden Folgezustände vermeiden will.

CONCLUSIONES

Las características anatomoquirúrgicas del exfínter anal han sido estudiadas de nuevo especialmente sobre la base de exámenes histológicos de la región anorrectal.

Con esto se ha comprobado que, además de los músculos ya conocidos como componentes del complejo muscular exfinteriano, es preciso también incluir la musculatis mucosae del ano y las músculos de los ligamentos anterior y posterior del exfínter.

Ha sido demostrado la relación entre la muscularis mucosae del ano y el exfínter interno con el septum intramuscular y con las glándulas.

Estas fibras submucosas fibroelásticas parecen ser el substractum de a las hemorroides internas y esto sugiere a la posibilidad de su papel en la patogenia de estas varicosidades.

Se ha llamado la atención sobre la importancia clínica del espacio intersticial, no descrito hasta ahora, que juntamente con los septums de la muscularis submucosa del ano cursan a través del exfínter intrno y con especial referencia a la propagación de las supuraciones anorrectales y de los tumores malignos.

La arquitectura del exfínter anorrectal es tal que el músculo exfínter tiene su músculo longitudinal asociado, lo que constituye una explicación anatómica de la inervación recíproca en la presentación de incontinencia anal consecutiva a las fistulotomías. La conservación de este mecanismo exfinteriano se considera de lo más importante para prevenir esta secuela, evitable, de la fistulotomía.

RÉSUMÉ

L'étude du caractère anatomique du mécanisme sphinctérien anorectal a été reprise du point de vue chirurgical sur la base d'examens histologiques.

II a étè mis en évidence qu'en plus des muscles habituellement mentionnés constituant cette structure composite, il est important d'inclure la tunique musculaire ainsi que les muscles des attaches antérieures et postérieures du mécanisme sphinctérien.

Les relations entre la tunique musculaire anale avec le sphincter interne, la formation de la cloison anale intermusculaire et les glandes anales sont démontrées.

II a été mis en évidence que ces fibres fibro-élastiques sousmuqueuses f o r m e n t le substratum des hémorroïdes internes, et il leur a été attribué un rôle probable dans la pathogénèse de ces varicosités.

L'importance clinque d'un espace interstitiel non décrit encore—l'interstice intermusculaire anal—avec les cloisons musculaires sous-muqueuses parcourant le sphincter interne est soulignée, particulièrement ence qui concerne l'extension d'une suppuration anorectale et les affections malignes. L'espace intermusculaire peut être considéré comme la porte d'entrée de la région périanale et de la fosse ischio-anale.

La structure du sphincter anorectal est telle que chaque muscle sphinctérien pos-

sède son muscle longitudinal respectif, ce qui fournit une explication anatomique à l'innervation réciproque et à l'apparition postopératoire de l'incontinence anale après fistulectomie. L'auteur insiste sur la préservation du mécanisme sphinctérien en tant qu'unité fonctionnelle anatomique lors de fistulectomie, séquelle iatrogen évitable.

RIASSUNTO

E' stato ristudiato il carattere anatomochirurgico del meccanismo dello sfintere anorettale in base ad esami istologici della regione anorettale. Si é dimostrato che, oltre ai muscoli noti che compongono questo complesso apparato, é necessario includere la muscularis mucosae dell'ano e i muscoli che si trovano in corrispondenza della parte anteriore e posteriore dello sfintere.

E' stato dimostrato un rapporto fra la muscolaris mucosae dell'ano e lo sfintere interno, il setto intermuscolare e le ghiandole anali. Queste fibre fibroelastiche submucose formano il substrato delle emorroidi interne e probabilmente hanno importanza nella patogenesi di tale affezione.

Viene messa in luce l'importanza clinica di uno spazio interstiziale, fino ad ora mai descritto, denominato interstizio anale intermuscolare, che ha uno speciale interesse nella diffusione delle suppurazioni anorettali e dei carcinomi. Questo interstizio deve essere riguardato come una via di passaggio alla regione perianale e alla fossa ischiorettale. L'architettura dello sfintere anorettale é cosiffatta che ad ogni muscolo sfinterico corrisponde un rispettivo muscolo logitudinale, associato ad esso, e ció spiega anatomicamente la innervazione reciproca e la comparsa dell'incontinenza anale post-operatoria dopo interventi di fistulectomia. E' molto importante, in tali operazioni, rispettare il meccanismo sfinterico, come unitá anatomo-funzionale, onde evitare questa temibile complicazione.

SUMARIO

As caracteristicas anatomicas do esfincter anoretal foram reinvestigadas e seu mecanismo considerado na base do esame histologico da região anoretal.

Establece que, alem dos musculos usualmente citados e importante incluir o musculo submucosa do anus e os musculos da inserção anterior e posterior do mecanismo esfincteriano. Demonstra as relações entre o musculo sub-mucoso e o esfincter interno para formação do septo anal intermuscular e as glandulas anais. Essas fibras submucosas fibrolasticas entram na formação do substratum das hemorroidas internas e assumem provavel papel na patogenese dessas varises. Esse espaço intersticial ainda não descrito—intersticio intermuscular—tem sido salientado especialmente com referência a difusão de supurações anoretais e neoplasias malignas. Considera o intersticio como uma porta de comunicação entre a região perianal e a fossa isquioanal. A arquitectura do esfincter anoretal é tal que cada musculo esfincteriano tem um conjugado muscular longitudinal, trazendo uma explicação anatomica para a inervação reciproca e o aparecimento de incontinencia nas fistulectomias. Essa sequela deve ser prevenida no curso das fistulectomias.

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The Publications Committee and Editorial Board

of the

Journal of the International College of Surgeons

have the honor to announce that

with this issue

Philip Thorek, M.D., F.A.C.S., F.I.C.S.

has assumed the duties of Editor-in-Chief of the Journal and Bulletin

Dr. Thorek succeeds his father, the late deeply lamented Dr. Max Thorek, Editor of the College publications since their inception.

Splenectomy for Myelofibrosis

ALFRED HAAS, M.D., F.I.C.S., AND GEORGE ROSENOW, M.D.

NEW YORK CITY, NEW YORK

THE concept of the syndrome characterized by splenomegaly, osteomyelofibrosis, hypoplasia of the bone marrow and a leukoerythroblastic blood picture (also called by many other names, e. g., chronic nonleukemic myelosis, agnogenic myeloid metaplasia and leukoerythroblastic anemia) has greatly changed in the last decade.

The case here presented, illustrates some practically important diagnostic, therapeutic and prognostic features of a still somewhat controversial condition.

Submitted for publication Sept. 16, 1959.

A case is reported in which an enlarged painful spleen was removed and the diagnosis of chronic myeloid leukemia was made on the basis of microscopic examination of the spleen and biopsy of the liver. Almost five years later this diagnosis had to be revised. Examinations of the marrow from various sites, roentgen studies of bones and bone biopsy established the fact that the patient had never had leukemia but had myeloid metaplasia of the spleen and myelofibrosis.

The important diagnostic procedures are emphasized, as is the fact that aspirations of bone marrow should not be limited to the sternum. The changed concept of the syndrome and its practical implications are discussed.

REPORT OF CASE

A 54-year-old white man had been followed at the outpatient department of a neighborhood hospital. The report of this clinic stated that the patient was a heavy drinker (4 or 5 quarts of beer a day) and that he had had gallbladder disease, cirrhosis of the liver and a diaphragmatic hernia. In the same clinic "macular erythematous lesions" were observed on his chest, abdomen, back and legs. The liver was palpable 3 fingerbreadths below the costal margin. There was no evidence of a diaphragmatic hernia on roentgen examination, but a large stone was noted in the gallbladder.

Laboratory data at the time were as follows: cephalin flocculation, 1 plus; serum bilirubin 1.9 mg. and total proteins 7.5 mg. per hundred cubic centimeters; albumin-globulin ratio, 5.7: 1.8. A blood count revealed 4,000-000 erythrocytes and 9,000 leukocytes per cubic millimeter, with a differential count of 68 per cent polymorphonuclear neutrophils, 30 per cent lymphocytes, 1 per cent eosinophils, and 1 per cent basophils. The urine was normal. The Mazzini test gave a negative result. A thoracic roentgenogram and an electrocardiogram revealed no abnormality.

The patient had been given a high protein, high carbohydrate, moderate fat diet, vitamins and rest. He discontinued his visits to the hospital clinic on June 9, 1954. One week later he was first seen by one of us (A. H.). He complained at that time of pain in the region of the gallbladder and still more pronounced pain over the left side of the abdomen. These "gas pains" were extremely severe, at that time barely tolerable.

Physical examination on June 15 revealed the patient to be 5 feet 3 inches (160 cm.) in height. He weighed 156 pounds (70.8 Kg.). The color of the skin and mucous membranes was normal. The temperature was normal. The blood pressure in millimeters of mercury was 160 systolic and 80 diastolic. The pulse rate was 60. The heart revealed regular no

sinus rhythm and no murmurs. It was slightly enlarged to the left. The lungs were clear. The tongue was not coated. There was no enlargement of the thyroid.

Abdomen: The liver, somewhat harder than normal, was palpable 3 fingerbreadths below the costal margin. The spleen was large and firm, reaching down to the umbilicus. On auscultation an extensive friction rub was heard over the splenic region. An abdominal flat film showed a solitary stone, about the size of a pigeon's egg, in the gallbladder.

For further observation and workup the patient was then admitted to Misericordia Hospital. The first blood count there revealed 3,220,000 erythrocytes and 10,250 leukocytes per cubic millimeter, with 11.8 Gm. of hemoglobin and a differential count of 63 per cent polymorphonuclear neutrophils, 31 per cent lymphocytes, 5 per cent monocytes, 1 per cent basophils and 405,000 platelets. The value for cholesterol was 188 mg. and that for serum bilirubin 2.4 mg. per hundred cubic centimeters. The icterus index was 14.7. In a fragility test, hemolysis started at 0.44 and was completed at 0.26.

Because of the gallbladder complaints and the tentative diagnosis of cirrhosis of the liver, it was decided to remove the gallbladder and to inspect the liver and examine it by biopsy. The nature of the splenomegaly was at that time not clear; the possibility of a relation to the suspected hepatic cirrhosis (precirrhotic enlargement of the spleen) was considered.

On June 30 cholecystectomy was performed. The gallbladder contained a solitary stone. The common duct was normal. The pancreas was without palpable induration. The liver was smooth, with no visible signs of cirrhosis. A liver biopsy specimen was taken from the gallbladder bed. A decision was made to remove the large perisplenitic spleen, which obviously caused great pain. There were many adhesions, but splenectomy was performed without difficulty in the usual manner. The spleen measured 34 by 12 by 14 cm.

The spleen and the biopsy material from the liver were sent to Dr. Paltauf (Misericordia Hospital, New York City) and material from the spleen to Dr. Neubuerger (General Rose Memorial Hospital, Denver, Colorado). The reports read as follows (only the pertinent contents are given):

Dr. Neubuerger: "The pattern of the spleen is obliterated. Only a limited number of small inconspicuous follicles are seen in several places; they are composed of apparently

Fig. 1.—Spleen, low power (× 75). Normal splenic structure almost totally obliterated. Only a few small follicles are visible.

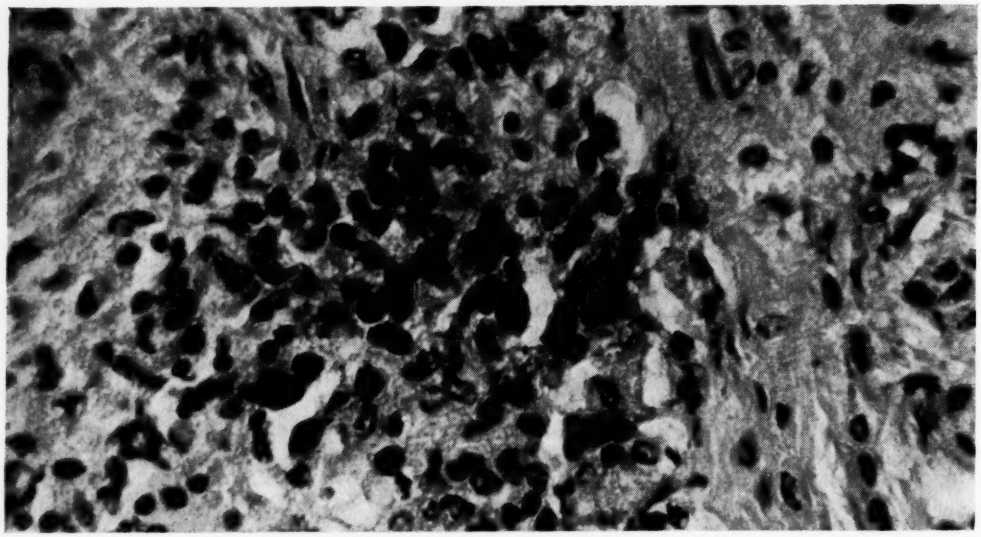

Fig. 2.—Spleen, high power (× 800). Red pulp is diffusely infiltrated with cells of myeloid series. Many normablosts are visible.

normal lymphocytes. Elsewhere there are no follicular structures. The red pulp is diffusely but rather unevenly infiltrated with pathologic cells. Most of these are large and have poorly defined, pink granular cytoplasm. Syncytial structures are frequent. In several spots the pathologic cells are quite densely packed and the nuclei have a tendency to be hyperchromatic. Other sections show a tendency to necrosis in the pathologic cell masses with loss of nuclear tangibility. It would appear that the pathologic cells are myelocytes or myeloblasts. Diagnosis: Hyperplasia, spleen, severe, myeloid leukemia" (See Figures 1 and 2.)

Dr. Paltauf: "The pulp of the spleen is congested and diffusely infiltrated with cells, which are apparently of myelogenous origin. The lymph follicles are small and contain few lymphoid elements. Sections of the liver show the normal architecture to be well preserved. In the periportal spaces and between the liver chords, infiltration by cells similar to those observed in the spleen are encountered. Diagnosis: Chronic cholecystitis; cholelithiasis; myelogenous leukemia of spleen? myelogenous leukemia of liver?"

The postoperative course was uneventful. Nevertheless, in view of the reports of the pathologists, deep concern arose, since splenectomy performed in the presence of myeloid leukemia has been disatrous in the majority of cases. Consequently it was expected that the patient would go downhill rapidly. Surprisingly, nothing of that kind happened. He continued to be well, resumed his work and at the time of writing (five years after removal of the spleen) is in fairly good health.

As the clinical course was clearly not consistent with the histologic diagnosis of leukemia, it appeared necessary to confirm or rule out this diagnosis in other ways, particularly by examination of the bone marrow. Unfortunately, this diagnostic test was not done prior to the operation.

Four months later (Oct. 11, 1954) a puncture aspiration of the sternal marrow showed a cellular hyperplastic marrow with normal distribution of nucleated white cells and an inversed red cell ratio amounting up to 60 per cent of all nucleated cells (Dr. J. Gudeman.). "The inverse ratio together with the enormous amount of nucleated red cells in the peripheral bloodstream and a marked increase in reticulocytes (up to 10 per cent) indicated a severe hemolytic process. Since the result of Coombs' test was negative, it was believed that an immunologic factor could be excluded."

At the same time definite anemia was observed. The value for hemoglobin was 68 per cent. There were 3,320,000 erythrocytes and 15,000 leukocytes per cubic millimeter, with 1 per cent myelocytes in the peripheral blood. Urobilinogen in the urine showed a pronounced increase.

The blood chemical data were then followed in the office up to the time of writing.

During the entire period there was always moderate to marked anemia: (July 7, 1955: erythrocytes, 2,900,000 per cubic millimeter of blood, with 10.8 Gm. of hemoglobin; leukocytes, 20,800 per cubic millimeter.) The number of platelets was constant at 350,000 to 400,000, and on one occasion (March 28, 1956) much higher (780,000). The blood smears always showed, during these years, a great number of nucleated red cells. Finally (January 1958), with the diagnosis still uncertain, the patient was referred to Dr. Rosenow. His observations and opinion follow:

When the patient was examined (more than three and one-half years after removal of the spleen) it was already apparent that the diagnosis of chronic myeloid leukemia, based on histologic studies of the spleen and liver tissues, had to be revised. The good general condition of the patient, after such a long period, was sufficient reason for that.

The numerous blood counts from 1954 to 1959 showed a constant tendency to subnormal values for hemoglobin and erythrocyte count and a pronounced increase in the number of white cells. Two illustrative examples are as follows. April 30, 1955: erythrocytes, 3,480,000 per cubic millimeter of blood; with 11.1 Gm. of hemoglobin; leukocytes, 19,050 per cubic millimeter, with a differential count of 62 per cent polymorphonuclear neutrophils, 27 per cent lymphocytes, 6 per cent monocytes and 5 per cent eosinophils. June 19, 1958: erythrocytes, 3,060,000 per cubic millimeter of blood, with 10.3 Gm. of hemoglobin; leukocytes, 35,000 per cubic millimeter, with 58 per cent polymorphonuclear neutrophils, 38 per cent lymphocytes and 4 per cent eosinophils.

A count made on Feb. 1, 1958 (A. H.) gave

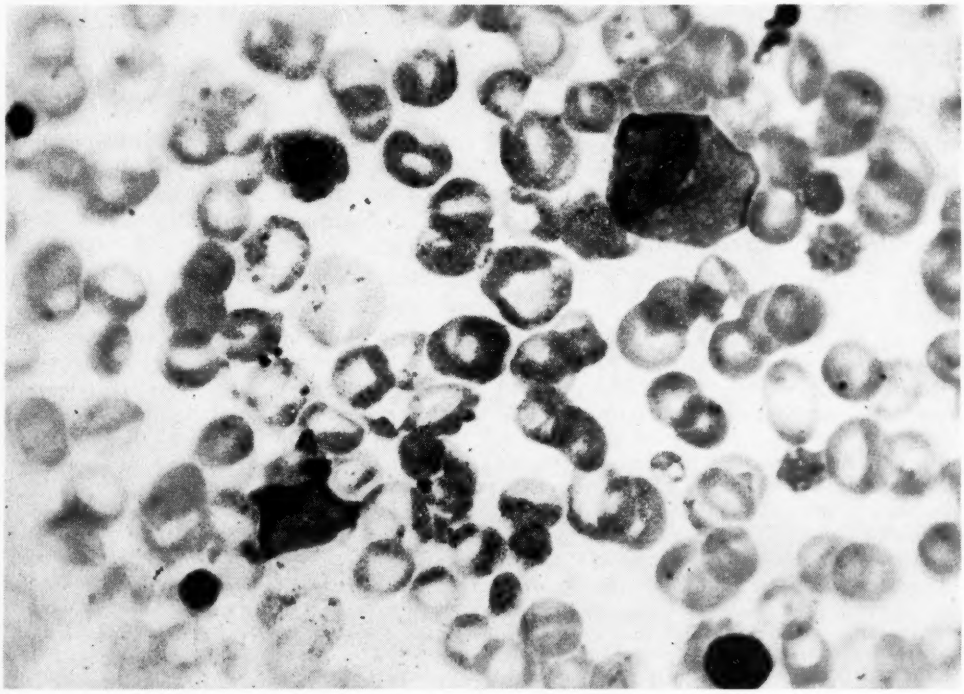

Fig. 3.—Blood, (peripheral) oil immersion (× 1600). Numerous normoblasts, one myelocyte and several Howell-Jolly bodies are visible in this field.

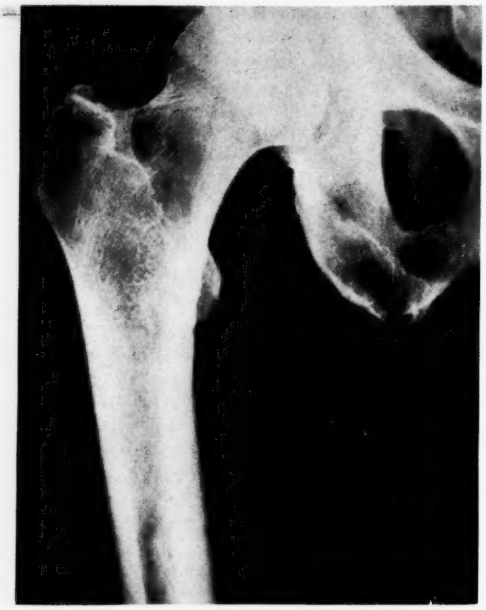

Fig. 4. — Upper third of right femur shows markedly thickened corticalis and osteofibrosis.

similar results: hemoglobin 9.3 Gm.; leukocytes, 30,000 per cubic millimeter of blood, with 55.5 per cent polymorphonuclear neutrophils, 4 per cent myelocytes, 32 per cent lymphocytes, 6.5 per cent monocytes and 1.5 per cent basophils. Most conspicuous was the large number of nucleated red cells, 309 nucleated red cells to 200 white cells. Almost all of these cells were normoblasts; a few pronormoblasts were observed. The corrected white cell count was therefore much lower, namely, 14,000 per cubic millimeter.

The morphologic character of the non-nucleated cells was abnormal. There was marked anisocytosis, with a number of spherocytes and a moderate degree of poikilocytosis with typical teardrop forms. Many of the erythrocytes were target cells, and a great percentage had basophilic stippling. Howell-Jolly bodies were observed in many erythrocytes (Fig. 3).

Attempts by one of us (A. H.) to aspirate bone marrow from both iliac crests yielded only a scanty amount of material, with few cells, insufficient for differentiation. The iliac bones were extremely hard. Sternal puncture, however, was easy, and ample material was obtained. The sternal marrow was cellular, with decidely increased erythropoiesis. The white series were normal; megakaryocytes were present in normal numbers. These observations of the marrow were strongly suggestive, *per se*, of myelofibrosis of the iliac bones.

This was substantiated by roentgenograms of the skeleton which showed marked osteofibrotic (sclerotic) changes in many bones, particularly the upper halves of both femurs, the bodies of the lumbar vertebrae and the iliac bones (Figs. 4 and 5). The definite anatomic evidence of osteofibrosis was finally provided by a specimen taken from the right iliac bone for biopsy (Fig. 6).

COMMENT

The aforementioned observations are evidence that the patient has, at present, myelofibrosis. Severe anemia, pronounced erythroblastosis in the blood and bone marrow and leukocytosis, with a shift to the left, are some of the prominent clinical features. Undoubtedly, the interpretation of the tissue change in the spleen and liver as diagnostic of chronic myeloid leukemia was erroneous.

Differentiation between myeloid leukemia and myeloid metaplasia, based solely on the study of microscopic sections, is extremely difficult and can even be impossible. The histologic data must be supplemented by examination of the blood and marrow. In the case here presented no signs of leukemia could be detected in either

Fig. 5.—Iliac bones show increased spotty density, indicating osteofibrotic foci.

Fig. 6.—Iliac bone biopsy specimen, low power (× 75). Note spotty increase of fibrotic tissue and small islands of bone marrow.

blood or bone marrow at any time after splenectomy. It appears reasonable to assume that myelofibrosis already existed at that time (i. e., about five years ago), although no roentgen examination of the bones was made before splenectomy or soon thereafter.*

The relation of myelofibrosis to myeloid metaplasia of the spleen has undergone thorough revision and reappraisal in the past ten years. Formerly, the prevalent opinion was that metaplasia of the spleen was secondary to myelofibrosis (or sclerosis) of the bone marrow. The myeloid metaplastic spleen was regarded as compensatory and as substituting for the deficient bone marrow. On the basis of such an assumption it appeared neither rational nor permissible to remove the spleen or to try to reduce its size by roentgen irradiation. Even modern text books on hematology stress this point of view. Whitby and Britton[1] expressed the opinion of many others when they wrote: "Splenectomy (i. e., in myeloid metaplasia) is valueless and usually fatal, as the enlargement of the spleen is compensatory and this organ is usually an important site of hematopoiesis." The concept expressed by Whitby is no longer generally accepted. The reasons are clinical as well as anatomic. An important clinical point against the "substitution" theory is the fact that patients with the syndrome in question

*The patient provided a roentgen film of the pelvis taken about twelve years earlier. No osseous changes are visible on this film.

survived splenectomy. Moreover, quite a number of these patients have actually been benefited by removal of the myeloid metaplastic spleen. Green, Conley and others[2] in a recent review of the literature and five cases of their own, conclude that splenectomy has no disastrous effects in these cases. Others, more recently Videbaek[3] and Dameshek,[4] have expressed analogous opinions.

Currently the syndrome of myeloid metaplasia of the spleen is considered a parallel manifestation of generalized myeloproliferative disorder (Dameshek,[5] Rohr,[6] Andreasen,[7] Fresen[8] and many others). Splenectomy for patients with the syndrome in question is no longer rejected summarily. Indications for the operation must be weighed carefully. Important points in favor of removal of the spleen are marked hemolysis or enlargement of the organ to such an extent that it causes mechanical difficulties, e.g. pressure symptoms as cited by Oechslin.[9] The peripheral "leukemoid" blood picture may be similar in both conditions.

In the presence of either condition, splenomegaly is a leading sign. The size of the spleen has no significance in differential diagnosis. Extremely large spleens are observed in patients with chronic myeloid leukemia and in those with myeloid metaplasia. On the other hand, leukemia with no enlargement or only slight enlargement of the spleen, is not too rare. Aspiration biopsy of the spleen will be of no great help in the differential diagnosis between leukemia and myeloid metaplasia.

Retrospectively, it is not easy to classify the case here described. No changes in the peripheral blood were reported prior to the operative procedure. Although neutrophilic leukocytosis is almost regularly observed after splenectomy (regardless of the underlying condition), the appearance of such great numbers of nucleated blood cells in the peripheral blood as was observed in our patient is uncommon.

The case shows clearly that the diagnosis between myeloid leukemia and myeloid metaplasia can be easily missed. Roentgen examination of the bones is not always helpful; early myelofibrotic foci may not show up on the films. Even more important is the fact that the myelofibrotic changes are unevenly distributed through the osseous system. Anatomic studies on extensive material have demonstrated that the early, spotty myelofibrotic tissue alterations start in the bones of the pelvis and the extremities; only at a later stage do the more central parts of the skeleton become involved in the process.[7] Other studies,[9] which have shown that osteomyelofibrosis is more pronounced in the iliac bones than in the sternum, have practical implications. They have shown the reason why puncture aspiration of the marrow solely from one site may be misleading. If only sternal puncture is performed the diagnosis of myelofibrosis can be easily missed. This happened in our case. When (shortly after splenectomy) a sternal marrow examination was done, no hint of myelosclerosis was detected. The sternal marrow was, at that time, highly cellular, and myelopoiesis and erythropoiesis at this site were very active. It is remarkable that almost 5 years later, cellular marrow was aspirated from the sternum again, whereas our repeated attempts to obtain marrow from the iliac crests were unsuccessful.

A few remarks should be made on the role of the spleen and the increased hemolysis in this case. It is doubtful whether the often loosely used term "hypersplenism" can be applied. One of the effects of splenectomy in the presence of hypersplenism is the decrease or disappearance of hemolytic manifestations. In our patient, removal of the spleen failed to accomplish this. The levels of serum bilirubin and

urobilinogen were unaffected. They still show a decided increase.

Attempts to explain the underlying mechanism would be entirely speculative. We are forced to admit that the manifold features of this case cannot be brought under one common denominator.

ZUSAMMENFASSUNG

Es wird über einen Fall berichtet, bei dem eine schmerzhafte vergrösserte Milz entfernt und auf Grund der mikroskopischen Untersuchung der Milz und einer Probeexzision der Leber die Diagnose einer chronischen myeloischen Leukämie gestellt worden war. Fast fünf Jahre später musste diese Diagnose revidiert werden. Untersuchungen des Knochenmarks aus verschiedenen Gegenden, Röntgenaufnahmen und Probeexzisionen der Knochen ergaben, dass der Patient niemals eine Leukämie sondern eine myeloide Metaplasie der Milz und eine Myelofibrose hatte.

Die wichtigen diagnostischen Verfahren werden hervorgehoben. Ansaugungen des Knochenmarks sollten nicht auf das Brustbein beschränkt bleiben. Die veränderte Auffassung des Syndroms und seine praktische Bedeutung werden erörtert.

RÉSUMÉ

Il s'agit d'un cas d'extirpation d'une rate douloureuse et augmentée de volume, où le diagnostic de leucémie myéloïde chronique a été posé sur la base d'un examen microscopique de la rate et d'une biopsie du foie. Près de cinq ans plus tard ce diagnostic a dû être revisé. Des examens de la moëlle à différents niveaux, des examens radiologiques osseux et des biopsies osseuses ont établi que le malade n'avait jamais eu de leucémie mais qu'il s'agissait d'un processus métaplasique myéloïde de la rate et de myélofibrose.

L'importance des techniques de diagnostic est soulignée. Les prélèvements de moëlle ne devraient pas être limités au sternum. La conception modifiée du syndrome, ainsi que ses conséquences pratiques sont discutées.

RESUMEN

Se trata del caso de un bazo voluminoso y doloroso en el que se hizo el diagnóstico de leucemia mieloide crónica sobre la base del examen microscáopico de la pieza operatoria y de la biopsia del hígado. Casi cinco años después este diagnóstico previo fué rectificado tras varios exámenes de la médula ósea tomada en varios sitios, el estudio radiológico de los huesos y la biopsia ósea, pudiéndose demostrar que el enfermo no había sufrido una leucemia sino una metaplasia mieloide del bazo y una mielofibrosis. Se resalta la importancia de los métodos de diagnóstico. La toma de tejido medular no debe limitarse al esternón. Finalmente el autor examina el concepto cambiado de este síndrome y sus aplicaciones prácticas.

SUMARIO

Comunicam um caso de esplenomegalia com resecção cirurgica e o diagnostico de leucemia mieloide cronica, comprovação histologica e biopsia do figado. Revisão esses diagnostico cinco anos depois. Exames da medula e de varios sitios, radiologia do esqueleto e biopsia de ossos demonstraram que o paciente nunca teve leucemia porem uma metaplasia melóide do baço com mielofibrose. Salientam a importancia desses diagnosticos. As biopsias—aspiração so devem ser feitas no esterno—Discutem a modificação de conceitos da sindrome e as deduções praticas advindas.

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In 1896, the year after the marriage of the Curies, a French scientist, Henri Becquerel, as previously mentioned, began his experiments which led to the discovery of what we now call radio-activity. The Curies were much interested in this new discovery, and Madame Curie set out to study the phenomenon with the aid of the instrument her husband and her brother-in-law had discovered several years before. The problem became so interesting that Pierre Curie gave up his studies on symmetry and joined his wife in this study.

An old abandoned machine-shop was turned over to them for use as a laboratory, and here they toiled. The source of uranium is pechblende, a costly mineral, mined chiefly in what is now Czechoslovakia, but which was, before the World War, Austria. In the manufacture of uranium a large quantity of refuse is formed, which they found was also radio-active. Through the intervention of the Academy of Science in Vienna, they obtained several tons of this refuse and began their efforts to extract the substance from it which produced these new rays.

After months of hard work the Curies finally extracted a substance which glowed in the dark and which they recognized as a new element—radium. Further study of this new element showed that it gave off, not one, but three kinds of rays, one group of rays resembling in many ways the X-rays of Röntgen.

—Major

Benign Stricture of the Esophagus Following Gastroenterostomy and Vagotomy

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AND

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IN the surgical treatment of chronic duodenal ulcer, gastroenterostomy and vagotomy as a combined procedure have, in recent years, evoked considerable interest and support. We here report an unusual, if not unique, case of benign stricture of the esophagus following transabdominal vagotomy and gastroenterostomy.

REPORT OF CASE

The patient, a farmer of asthenic constitution aged 59, was admitted to the Regional Hospital, Galway, in November 1957. Gastroenterostomy and vagotomy had been performed four years earlier for chronic duodenal ulceration. This operation had given a satisfactory result, with complete relief from peptic ulcer symptoms. The patient stated that for the twelve months preceeding his admission (November 1957) there had occurred transient episodes of mild difficulty in swallowing. Three days prior to admission the dysphagia had suddenly become severe, and he had been unable to retain any solid or liquid food since the onset of this attack. He felt a "holdup" at the level corresponding to that of the xyphisternal joint.

On admission the patient was grossly dehydrated but otherwise in good health. The dehydration was corrected, and he was maintained for six days on intravenous fluids, after which time he was able to retain a high caloric fluid diet. A barium swallow and meal revealed a stricture of the lower end of the esophagus, which was considered due to fibrosis. A well-functioning gastroenterostomy was also demonstrated.

Esophagoscopic study confirmed the presence of the stricture; the mucous membrane above the level of the stricture was ulcerated, and biopsy of this area revealed no evidence of malignant disease.

Four days later the stricture was endoscopically dilated by bougies. This dilatation was carried out with difficulty and the stricture gave the impression of being very tight.

It was now Christmas, and as the patient could take a semisolid diet with comfort he was allowed to return home.

He was readmitted on January 30; he was then in poor condition and again could take only a liquid diet. A barium meal showed the stricture to be tighter than at the previous examination, and by the esophagoscope it was possible to pass only the finest bougie (Fig. 1).

Owing to the extreme tightness of the stricture, a thoracotomy for its excision was considered the safest procedure, or possibly dilation under direct vision. At operation the stricture was located about 3 inches (7.5 cm.) from the cardiac orifice. It was short, involving about ½ inch (1.2 cm.) of the esophagus, but a small hard lump, yellowish white, could be palpated on the left antero-

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The authors describe a case of dysphagia occurring some years after vagotomy for duodenal ulcer and suggesting stenosing esophagitis, in which the cause proved to be a neuroma at the point of section of the vagus, similar to an amputation neuroma.

lateral surface. This tumour was dissected free and proved to represent the enlarged and indurated end of the vagus. It was firmly adherent and partially surrounded the wall of the esophagus. Bougies up to the maximum size—No. 28 French—were then passed with ease. No further procedure was carried out, and the chest was closed with seal drainage. Postoperative progress was satisfactory when the patient was discharged, on March 21. There has been no recurrence, and he can swallow any kind of food.

Pathologic Report.—The report on the tissue removed was as follows: "Foreign body granuloma due to suture material present. Proliferated nervous and fibrous tissue present (Fig. 2)." It was not possible to arrange the patient's return to our follow-up clinic, but in recent correspondence with his physician (January 1959) we are informed that he is in excellent health, with no dysphagia, vomiting or other gastrointestinal symptoms.

Fig. 1.—Preoperative barium swallow, February, 1959.

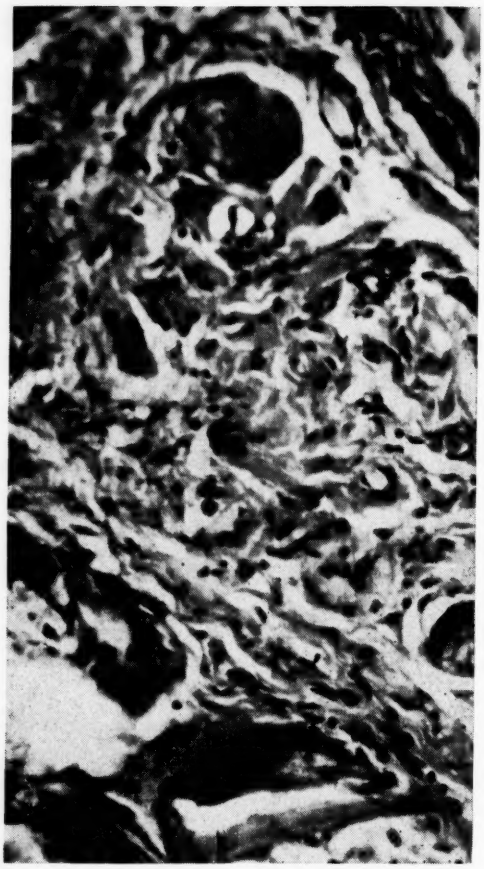

Fig. 2.—Foreign body reaction to suture material with fibrosis. (Hematoxylin and eosin: x 400.)

Up to the date of investigation of this case our customary technic in performing vagotomy has consisted of traction on the lower end of the esophagus, ligation of the mobilized strands of the vagi with cotton thread, and then their division distal to the ligature. This meant that the proximal stump and ligature were retracted into the thorax.

The stricture in this case resulted from inflammatory reaction to the ligature material, combined with a small neuroma of the proximal end of the divided left vagus

nerve. It is now our custom to ligate the vagi flush with the stomach wall and to leave the upper ends untied.

Author's Note: We wish to express our gratitude to Prof. J. Kennedy and Dr. W. Bennett of the Pathology Department for their assistance in the preparation of this report.

ZUSAMMENFASSUNG

Die Verfasser beschreiben einen Fall von Schluckbeschwerden, die einige Jahre nach einer wegen Zwölffingerdarmgeschwürs ausgeführten Vagotomie auftraten und an eine stenosierende Speiseröhrenentzündung denken liessen. Die Ursache der Erkrankung wurde in einem Neurom an der Stelle der Durchschneidung des Vagus gefunden, das einem Amputationsneurom ähnelte.

RESUMEN

Describe el autor un caso de fisfagia aparecida algunos años despues de una vagotomía por úlcera duodenal y cree pueda tratarse de una esofagitis estenosante provocada por un neuroma en el punto de sección de un nervio vago semejante a los neuromas de amputación.

RÉSUMÉ

Les auteurs décrivent un cas de dysphagie quelques années après vagotonie pour ulcère duodénal qui ressemblait à une oesophagite sténosante, mais où ils ont trouvé comme cause un neurome au point de section du pneumogastrique, comparable au neurome d'amputation.

RIASSUNTO

Ofli autori describono un caso di dysphagia qualchi anni dopo una vagotomia por ulcus duodenale, chi rassomiglió a una oesophagitis stenosous ma nel quale si trovó all'operazione como causa un neuroma al punto di sezione del vago, comparabile ad un neuroma di amputazione.

SUMARIO

Descreven um caso de disfagia após alguns anos da vagotomia para ulcera duodenal e sugerem tratar-se de esofagitee estenosante causada por um neuroma no ponto de seçcão do vago, como ocorre nos casos de amputação.

Although he could not introduce new treatment, William Jenner gave countenance to the forces of progress in the grim battle which was going on all around him in the field of sanitation, and which is described in the next chapter of this book. But Jenner was the authentic physician, that is a man devoted to the actual patient and to analysing how his illness differed from all other cases. Like Melampus, he believed in the daily vigil, keeping the honest record of every sound nature uttered, joining letter to letter, word to word, until he could at last spell out nature's meaning.

—*Williams*

Genitourinary Surgery

Conservative Management of Renal Injuries

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DIRECT force is the usual cause of injury to the kidney, whether the resulting wound is penetrating or nonpenetrating.

The degree of force required to produce a renal injury need not necessarily be high. It is this fact which makes the following case presentations worthy of consideration.

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Three cases of renal trauma are presented. In the first, trauma was the result of an automobile accident with sufficient force to cause rib fractures. In the second it was caused by compression of the loin from a heavy object and in the third the patient was a child who had fallen.

Gross hematuria was the outstanding sign, and roentgenographic evidence established the extent of the injury. Conservative management, consisting of bed rest and administration of intravenous fluids, with or without the transfusion of whole blood and antibiotics, and continuous observation are imperative. Blood pressure readings are taken every hour if necessary. An erythrocyte count is made and a urinalysis performed daily until the clinical course becomes stabilized. Follow-up roentgenograms and observation are advisable during the ensuing year.

During the past wars, those of us who were fortunate enough to have a Urologic Service in one of the military hospitals saw may injuries to the kidneys due to penetrating wounds. They were associated with penetrating wounds of the abdominal viscera, usually caused by shell fragments, bullets and stabs. The kidneys were repaired or removed during the course of general abdominal surgery. Similarly, we saw many blast injuries, when the patient had been subjected to tremendous external force due to an explosion, and again abdominal and renal surgical treatment was life-saving.

The symptoms of renal injury are primarily shock, pain, hematuria and a mass in the loin.

Civilian practice brings the surgeon into contact with renal injuries sustained by less violent means, and conservative management is more commonly employed. These patients are seen hours after the accident. They have grossly bloody urine, and some discomfort or pain in the flank; they are not in shock, and there is adequate time for examination and evaluation.

Pain in such patients usually is not due as much to the injury of the kidney as to injury of muscles of the abdomen and chest, with or without fracture.

Gross hematuria is usually the symptom that brings the patient to the hospital. Hematuria, however, may be microscopic,

and catheterization may be necessary to obtain a specimen.

The patients whose cases are here reported did not have an extreme degree of shock. Hematuria was the presenting sign, and tenderness to palpation of the loin was more prevalent than was a mass.

Examination of a patient suspected of having renal injury should include an immediate scout film of the kidney-ureter-bladder area. This film will quickly identify the kidneys, and some estimate of size, shape and position may be made. Irregularity of the renal outline is frequently noted, and any filling of the loin or production of a mass can be seen. One should look for signs of ileus and free gas in the peritoneal cavity.

Whenever possible, an intravenous urogram should be obtained, since urography better evaluates the damage and enables one to make some estimate of renal function. Extravasation of urine may be suspected and in some cases is visualized as the contrast medium appears to go beyond the confines of the calyceal system.

Cystoscopic and retrograde pyelographic examination should be done whenever feasible. With present-day aseptic technic there is little risk, and the retrograde pyelogram will give the examiner more complete information.

REPORT OF CASES

Case 1—R. G., a 28-year-old white man, was admitted from the emergency room immediately after being struck by an automobile.

On physical examination he was coherent and fairly comfortable. There were abrasions of the head and face and contusions of the left thoracic cage and the left flank, with possible rib fractures and left renal trauma. The systolic blood pressure was 130 mm. of mercury, the diastolic 80 mm. The temperature 100F; the pulse rate 88, and the respiratory rate, 18, per minute. There was pain on motion. The erythrocyte count was 5,000,000 per cubic millimeter of blood, with 80 per cent (12.5 Gm.) of hemoglobin. Urinalysis revealed cloudy brown urine with a specific gravity of

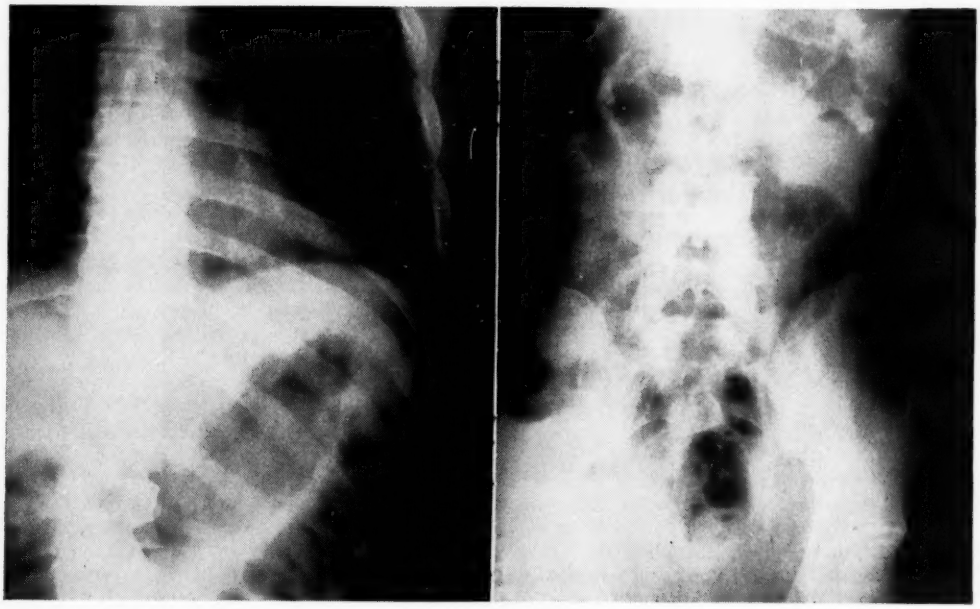

Fig. 1. (see text).

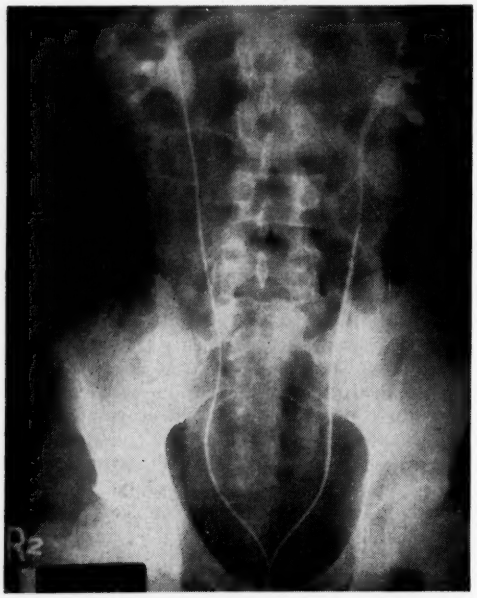

Fig. 2 (see text).

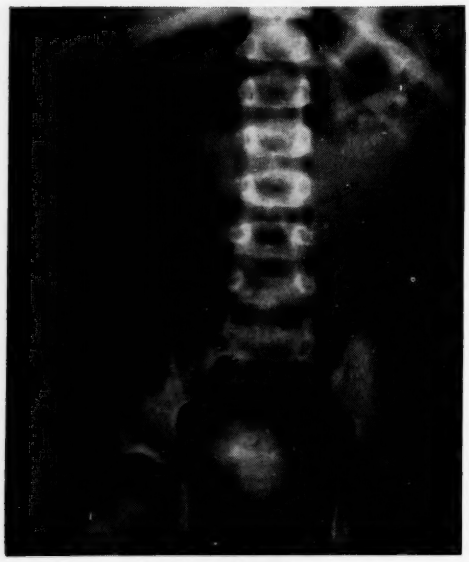

Fig. 3 (see text).

1.027, containing 60 mg. of albumin. An occasional pus cell and 20 to 22 erythrocytes per high powered field were observed. Urinalysis performed later on the day of admission showed 750 mg. of albumin, and microscopically the urine was loaded with erythrocytes. The value for blood urea nitrogen was 11 mg. per hundred cubic centimeters.

Roentgenographic examination revealed transverse complete fractures of the ninth, tenth and eleventh ribs in the midaxillary line on the left side. A flat plate of the abdomen revealed some degree of ileus, with distention of both the small and the large bowel. The psoas shadow was obscured, and the left kidney shadow enlarged.

An intravenous urogram taken at this time showed prompt excretion of the contrast medium from both kidneys. The right upper portion of the urinary tract presented a normal appearance. On the left there was some dilatation of the pelvis and calyceal system, and contrast medium was noted in the parenchyma, indicating traumatic rupture of the kidney.

The patient was treated with absolute bed rest, intravenous fluids, urinary antiseptics and a moderate diet; the fractures were treated by immobilization.

Hematuria slowly diminished until the tenth day, when only an occasional erythrocyte was visible microscopically.

Eighteen days after the day of the accident cystoscopic and retrograde pyelographic studies were performed. At this time observations were well within normal limits. The renal structures presented a normal appearance, and no contrast medium was noted in the renal parenchyma. Urine was collected from both kidneys, and cultures failed to reveal any growth of bacterial organisms.

The patient was discharged after twenty-nine days of hospitalization.

Case 2—J. J., a 5-year-old white boy, was admitted to the Urologic Service from the emergency room after an injury to the abdomen. This history is unusual, in that the child had been attempting to climb into an empty refrigerator. Everyone has read many accounts of children who died from suffocation in a refrigerator; this child's experience, however, was somewhat different. The refrigerator door was open, and as the child attempted to climb into it the box tilted and fell upon him. A mattress lying on the floor broke the fall, and, the door being open, he was struck by the upper shelf of the refrigerator across the abdomen.

Shortly after the accident the child complained of severe abdominal pain and had gross hematuria. He vomited once before being

brought to the hospital. Examination of the abdomen revealed guarding and splinting of the left upper quadrant, with subjective pain on palpation of the left costovertebral region. No masses were palpated, and minimal peristalsis was heard. Right costovertebral tenderness was absent.

The urine at this time was brown and very cloudy, with a specific gravity of 1.021. It contained 400 mg. of albumin per hundred cubic centimeters and microscopically was loaded with erythrocytes. The value for blood urea nitrogen was 18 mg. per hundred cubic centimeters. A blood count showed 3,300,000 erythrocytes per cubic millimeter, with 58 per cent (9 Gm.) of hemoglobin. The hematocrit reading was 31 volumes per cent. The systolic blood pressure on admission was 126 mm. of mercury and diastolic 84 mm; the pulse rate, 116 per minute.

The clinical picture remained unchanged during the night, and intravenous urographic examination was performed. After the intravenous injection of the contrast medium there was prompt excretion from the left side with distortion of the calyces. At ninety minutes

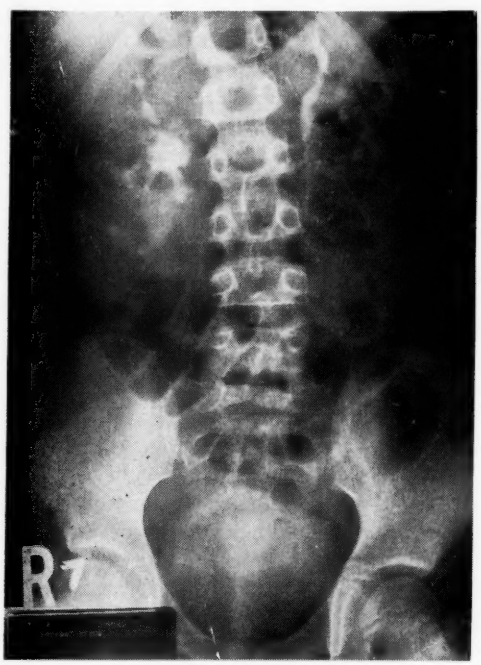

Fig. 4 (see text).

Fig. 5.—Roentgenograms taken ten months after injury revealed some residual deformity of pelvocalyceal system, but there was no extravasation of contrast medium and no hematuria microscopically. Infection described previously had subsided and culture of urine gave negative result.

there was some collection of the contrast medium in the lower portion of the left kidney, due to extravasation into the renal parenchyma. The outline of the kidney was not well delineated. The left ureter appeared normal. There was no evidence of a renal shadow on the right side.

(There was no abnormal distention of the small or large bowel, and the psoas shadows were not well delineated.) The child's temperature rose to 102.4F. twenty-four hours after admission and "spiked" daily for four days. During this time antibiotic therapy, intravenous fluids and 250 cc. of whole blood were administered.

After five days of bed rest the patient had less abdominal pain; the temperature returned to normal, and he was less restless. After eight days the abdomen was soft and there was only slight tenderness in the left upper abdominal quadrant. By the twelfth day the patient was completely afebrile and the abdomen was soft

and not tender; hematuria, however, persisted.

Cystoscopic examination was performed. There was no evidence of a ureteral orifice on the right side; nor was the right side of the trigone developed, which confirmed the diagnosis of solitary left kidney with laceration.

Microscopic hematuria continued in this patient but was so slight that on the twenty-seventh day he was discharged with instructions for restricted activity to be followed in the clinic. One month later the urine cleared and normal results were obtained from urinalysis.

Case 3—D. D., a 5-year-old girl, was admitted to the Urologic Service from the emergency room. The child stated that she had been running across the street, playing and attempting to race her older sister, when she fell in the snow, striking her abdomen against the curb at the sidewalk. She did not notice too much pain when she fell, since she was wearing a very heavy snowsuit. It was not until she arrived at home and urinated that the parents appreciated the seriousness of the injury. The urine was grossly bloody, and the parents brought the child to the hospital at once.

A roentgenogram of the abdomen revealed enlargement of the right kidney and scoliosis of the lumbar portion of the spine, with a convexity to the right. There was no evidence of free air in the abdominal cavity, but there was some degree of ileus of the small bowel.

The systolic blood pressure was 110 mm. of mercury and the diastolic 70; the pulse was full and strong. The erythrocyte count on admission was 3,700,000 per cubic millimeter of blood, with 70 per cent hemoglobin (11 Gm.). The value for blood urea nitrogen was 15 mg. per hundred cubic centimeters. The urine at this time was dark brown and cloudy: the specific gravity was 1.032. There were 400 Gm. of albumin per hundred cubic centimeters. Microscopically, the urine showed innumerable erythrocytes.

The intravenous urogram taken on admission did not outline the psoas muscles. There was no evidence of fracture of the ribs or the lumbar vertebrae. After intravenous injection of the contrast medium there was prompt excretion on both sides. The left upper portion of the urinary tract appeared entirely normal. The right showed the extravasation of contrast medium outside the calyceal system into the renal parenchyma, and there was a suggestion that some of the contrast medium was extravasated into the subcapsular space. Both kidneys drained rather promptly in the erect position. There was retention of the contrast medium, however, in the parenchyma where the extravasation existed.

A culture of the urine revealed Micrococcus aureus, which was treated by antibiotic therapy.

In twenty-four hours there was a drop in the hemoglobin level to 67 per cent (10.5 Gm.). The hematocrit reading was 38 volumes per cent. Intravenous fluids and the transfusion of whole blood were followed by an erythrocyte count of 4,000,000, with 12 Gm. (76 per cent) of hemoglobin. The leukocyte count was never elevated above 10,250 per cubic millimeter.

For the first forty-eight hours after admission the clinical course was stormy with considerable abdominal pain, distention and rigidity. There was elevation of the temperature, by midnight of the second day, to 103F. This continued by spiking (100 to 102F.) for four days.

Surgical and Pediatric consultations during this period were utilized, and the advice of both consultants confirmed the urologic opinion that conservative management should be continued. There was no evidence of abdominal visceral injury. Twenty days after admission the urine failed to reveal any continuation of microscopic hematuria, and the patient was discharged from the hospital for continued observation at home.

RESUMEN

Se presentan 3 casoss de trauma renal. En el 1º el trauma se produjo en un accidente de automovil que determinó rotura de costillas. El segundo consistió en compresión de la región lumbar por un objeto pesado y el tercero fué debido a una caída de un niño jugando.

El signo predominante fué la hematuria masiva y ardiográficamente se pudo establecer la extensión de la lesión.

El tratamiento conservador es fundamental, es decir: reposo en cama, fluidoterapia intravenosa, con o sin transfusión de sangre, antibióticos y observación continua. Si es preciso debe medirse la presión sanguínea cada hora. A diario, y hasta que el curso clínico se estabilice,

deben investigarse el cuadro hemático y la orina.

Durante un año es aconsejable la observación periódica con exploración radiográfica.

ZUSAMMENFASSUNG

Es wird über drei Fälle von Nierenverletzung berichtet. Im ersten Falle handelte es sich um einen Automobilunfall, der schwer genug war, um Rippenbrüche zu verursachen. Im zweiten Falle bestand die Verletzung in einer Quetschung der Lendengegend durch einen schweren Gegenstand, und im dritten Falle handelte es sich um ein Kind, das beim Spiel gefallen war.

Das hervorstechende Krankheitszeichen war schwere Harnblutung, und der Umfang der Verletzung wurde im Röntgenbilde nachgewiesen.

Konservative Behandlung ist unbedingt notwendig und besteht in Bettruhe und intravenöser Verabreichung von Flüssigkeiten mit oder ohne Bluttransfusionen, in der Verabfolgung von Antibiotika und ununterbrochener Beobachtung des Patienten. Wenn nötig, werden stündliche Messungen des Blutdrucks vorgenommen. Zählung der roten Blutkörperchen und Harnuntersuchung erfolgt täglich, bis der klinische Ablauf sich stabilisiert.

Weitere Beobachtung mit Röntgenkontrollen empfiehlt sich für die Dauer eines Jahres.

RÉSUMÉ

Trois cas de traumatismes des reins sont présentés: 1. accident de voiture suffisamment violent pour provoquer des fractures de côtes; 2. forte compression lombaire; 3. chute en jouant (enfant).

Le signe le plus marquant a été une forte hématurie. La radiologie a mis en évidence l'extension des lésions.

Le traitement qui s'impose est conservateur: repos au lit, liquides intraveineux, avec ou sans transfusions de sang complet, antibiotiques, surveillance ininterrompue. La pression sanguine est contrôlée toutes les heures selon les cas. Numération des érythrocytes et analyse d'urine tous les jours jusqu'à stabilisation de l'évolution clinique.

Il est recommandé de continuer à suivre ces cas pendant encore une année environ et de pratiquer des contrôles radiologiques.

RIASSUNTO

Vengono presentati tre casi di trauma renale. Nel primo il trauma era dovuto ad un incidente automobilistico e vi erano fratture costali; nel secondo vi era stata una compressione della regione lombare da parte di un oggetto pesante e il terzo era un bambino che era caduto mentre giocava. Il segno clinico dominante era l'emorragia; con l'esame radiologico si mise in evidenza l'estensione della lesione.

E' indicata la cura consevativa, consistente nel riposo a letto e nella somministrazione di liquidi endovena con o senza trasfusione di sangue. Se é necessario si deve controllare la pressione ogni ora; la conta dei globuli rossi e l'esame di urina si praticano una volta al giorno fino alla stabilizzazione del processo.

E' consigliabile fare controlli clinici e radiologici nell'anno successivo al trauma.

SUMARIO

Apresenta tres casos de traumatismo renal. No. 1º caso houve acidente de automovel e fratura de costelas. No 2º caso compressão da coxa e no 3º queda de uma criança durante um jogo. Hematuria macissa foi o grande sinal e a radiologia evidenciou a lesão. Realisou o tratamento conservador, repouso, hidratação, antibioticos e vigilancia. Mostra a sequencia controlada por estudo clinico e pela radiologia durante um ano.

Neurologic Surgery

Coccygodynia

An Analysis of One Hundred Cases

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BECAUSE coccygodynia often occurs after long periods of watching television, I am inclined to coin the term "television bottom" for this condition. Coccygodynia has been atttributed to neurosis, neuritis, neuralgia, rheumatism, strain, luxation and fracture. Treatment has included sedatives, antineuritics, antispasmodics, rectal suppositories, hot and cold applications, electrotherapy, massage of the coccyx, radiation therapy and even coccygectomy.

Patients with coccygodynia, or "television bottom," complain of pain in the coccyx and adjacent regions. Thiele,[1] however, has shown that in about 85 per cent of patients the pain occurs not in the coccyx but in either the sacrococcygeal joint or the muscles (the levator ani, the coccygeus and a bundle of fibers from the gluteus maximus) that are partially inserted into the lateral borders of the coccyx.

From the Lexington Clinic, Lexington.
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Pain in the coccygeal region is discussed and its diagnosis and treatment described. The various causes are listed, and it is pointed out that in some instances it is due to sitting overlong before the television set, especially if the posture is poor. Infection may also be responsible, requiring surgical removal of its focus.

Typical Case.—Patients with coccygodynia characteristically sit down cautiously, then repeatedly shift their position from one side to the other. Many wish to stand while their histories are being taken. Women seem to be more susceptible than men to this syndrome. Most of the women note exacerbation of their symptoms immediately before and during the menstrual period.

Almost without exception, the patient says that a long automobile ride or prolonged sitting will cause severe pain. He definitely says that he cannot lie in bed comfortably on his back at night. The patient habitually sits in poor posture, with the lumbosacral portion of the back convex instead of concave. He slumps in a chair and allows the middle portion of the sacrum and coccyx to press against the chair. He usually sits down in and gets up from a chair slowly and carefully.

Examination.—A patient may be examined in the left lateral Sim's position or in an inverted position. The examining index finger is inserted full length into the rectum. The finger is placed over the

coccyx internally and the thumb over the coccyx externally. The coccyx is palpated and moved anteriorly, posteriorly and laterally. In most cases of coccygodynia a movement of the sacrococcygeal joint produces severe pain. In some cases, however, according to Thiele,[1] the joint can be moved without pain, although there are still severe tenderness and spasm of the levator ani and coccygeus muscle. Normally the sacrococcygeal joint is freely movable, the anteroposterior flexion and extension of the coccyx in relation to the sacrum being about 30 degrees. Thiele adds: "The tip of the coccyx may be normally moved laterally about 1 cm. from the midline. Mobility of the coccyx is therefore not a sign of dislocation or fracture. If a fracture is present, any movement of the bone is extremely painful and crepitation may be felt. An x-ray will confirm the diagnosis."

After the coccyx is palpated in the examination as afore described above, the examining finger is moved laterally from the posterior midline, with the palmar surface in contact with the soft tissues. The medial borders of the gluteus maximus, the levator ani and the coccygeus are felt as they proceed to their point of insertion into the lateral edges of the coccyx and the lower portion of the sacrum. With moderate pressure, the finger is moved laterally, anteriorly and then medially through 180 degrees, until it lies just posterior to the symphysis pubis. Thus, one can palpate all of the fibers of the muscles that are inserted into the lateral border of the coccyx.

Thiele has stated: "The most typical and characteristic findings in coccygodynia are muscle tenderness and spasm." The coccygeus muscle is more difficult to assess because of an underlying ligament, and may be thought to be in a state of spasm when it is not. When the levator ani is spastic, it is felt as a firm sheet of muscles stretched from its origin in the obturator fascia to its insertion in the lateral borders of the coccyx and lower portions of the sacrum. One often can feel this as a sort of "bowstring" stretching across this area. If the patient strains downward, the spastic levator muscles will often relax while being palpated. One should remember that this muscle spasm and tenderness may be unilateral or bilateral. If it is unilateral the stress on the sacrococcygeal joint is in the direction of flexion and abduction, and the pain is often more severe on that side. If bilateral, it is in the direction of anterior flexion, according to Thiele.

After digital examination of the rectum, complete proctoscopic and anoscopic examinations should be performed to detect any coexisting pathologic conditions.

In the male, the prostate and seminal vesicles should always be palpated. A pelvic examination should be performed on a woman.

History.—The patient's history is of great value in diagnosing this condition. In a history characteristic of coccygodynia the pain is definitely exacerbated by continued sitting, is most severe while rising from a sitting position, and is aggravated by long motor rides.

A few exceptions to this general rule are patients in whom pain is due to severe trauma or fracture, or to rare conditions such as a tumor of the spinal cord or herniation of an intervertebral disc. In cases of severe trauma or fracture the pain is increased during defecation. If sudden trauma has been the causative factor, muscle spasm occurs at once, and the onset of pain and tenderness is immediate.

Patients often say that pain in the hip and leg accompanies the pain in the tail bone. It is caused by pressure of an associated spastic piriformis muscle on the sciatic nerve as it emerges through the sacrosciatic foramen and by pressure upon the superior gluteal nerve as it passes be-

tween the upper border of the spastic piriformis and the lower border of the gluteus medius.

Approximately one-fourth of my patients complained of associated pain in the upper portion of the gluteal region or down the posterior portion of the thigh. Examinations of these patients revealed a spastic piriformis muscle.

Freiberg and Vinke[2] and Freiberg[3] have published two excellent papers discussing the relation of piriformis muscle spasm to sciatica from an orthopedic standpoint. Freiberg's sign of piriformis muscle spasm is limitation of motion on inward rotation of a fully extended thigh.

Etiologic Factors.—In Thiele's opinion,[1] coccygodynia is due to three definite factors: (1) Direct trauma; (2) continued immobilization of a joint in a position of flexion by muscle tension, and (3) muscle spasm. With regard to point 2, in cases of coccygodynia the sacrococcygeal joint is maintained in a state of constant flexion by tension of the spastic levator ani and coccygeus muscles. Says Thiele[1]: "We are taught that repeated or sustained muscle contraction results in the formation of lactic acid, which causes fatigue and then pain. Anoxia may also be a factor."

Poor posture may produce muscle spasm if continued if continued over long periods. The spasm is due more to a continued overflexion of the sacrococcygeal joint than to direct pressure on the coccyx.

Table 1 lists the etiologic factors in my series and the number of patients in each category. Myositis or reflex spasm of the levator ani, coccygeus and piriformis muscles may be caused by a focus of infection when the lymphatic drainage is toward the pelvic muscles. In my series, infection was the etiologic factor in 60 cases (Table 1). The infection was discovered in the prostate in 3 cases, in the epididymis in 1, in the cervix in 5 and in the anal region in 51.

In 38 cases trauma was thought to be the etiologic factor. One can easily elicit a history of some kind of fall in most cases, but 18 of my patients had definite history of a fall severe enough to cause direct trauma to the coccyx. There were no fractures. Three patients were pregnant when they fell. In 14, prolonged sitting while watching television was definitely thought to be the traumatic factor. In 3 cases prolonged automobile rides seemed to be responsible.

Two patients complained of a dull, heavy aching in the lower part of the rectum and the sacrococcygeal area a few weeks after an anorectal operation. This pain was severe enough to be placed in the category of coccygodynia that developed post-operatively.

Method of Treatment.—As early as 1910 Ely[4] was massaging the coccyx and its immediate soft parts between his thumb and finger. He said that "usually two or

TABLE 1.—*Etiologic Factors*

Infection	60 cases
Prostatic	3 cases
Epidydimal	1 case
Cervical	5 cases
Anal	51 cases
Trauma	38 cases
History of fall	18 cases
Fracture	0 cases
Pregnant at time of fall	3 cases
Prolonged sitting	14 cases
Prolonged rides	3 cases
Postoperative	2 cases
No spinal cord tumors and no herniated disc	
Total	100 cases

TABLE 2.—*Methods and Effectiveness of Treatment*

Treated by massage only	62 cases	
Relieved of symptoms	50 cases	(80%)
Unrelieved of symptoms	12 cases	(20%)
Treated by instructions in regard to posture and heat (dry heat or hot baths or both)	28 cases	
Relieved	25 cases	(90%)
Unrelieved	3 cases	(10%)
Treated by massage and surgical removal of foci of infection	10 cases	
Relieved	9 cases	(90%)
Unrelieved	1 case	(10%)
Total	100 cases	

three treatments at intervals of two or three days would suffice to cure the cases."

Thiele[1] has had more experience and contributed more to the literature on coccygodynia than has any other author. He has described a very effective method of massage, which a great number of proctologists have adopted. Here is his description of the technic: "With full-length insertion of the finger into the rectum latero-posterior pressure will place its flexor surface horizontally across the surfaces of the levator ani and coccygeus muscles almost at a right angle to their fibers. The fibers of the piriformis are felt immediately beyond the sacrospinus ligament and are touched by the finger tip in such a manner that lateral motion of the finger will stroke lengthwise that portion of the belly of the muscle lying within the pelvis.

"These muscles are massaged in the long direction of their fibers and in the same manner as a strop is stroked by a razor. Massage is begun lightly. This is necessary because one does not wish to traumatize the extremely tender spastic muscles. The sacrospinus ligament is merely pressed on by the finger in the direction vertical to its long axis. As the patient makes subsequent visits, massage is made with increasing pressure. If a reaction which is evidenced by increased pain is evoked, light massage is again reverted to and pressure is increased as tenderness decreases.

"If definite improvement does not result after the first four to six massages over a period of a week or ten days, orthopedic or other indicated consultation should be sought."

Most of the patients in my series were treated by massage and were given an average of six treatments over three to four weeks.

Medical and surgical control of foci of infection was used when necessary. This is reported in the results and consisted of removing chronic anal fissures, hemorrhoids and fistulas when they were deemed a part of the condition and the pain was not relieved by massage.

I have not performed a coccygectomy and mention it only to condemn the operation as a cure for coccygodynia.

Results of Treatment.—As Table 2 shows, 62 patients were treated by massage only, and symptoms were relieved in 80 per cent, or 50 patients. Twenty per cent, or 12 patients, were not relieved by massage.

Twenty-eight patients were treated by careful instruction in proper posture, and by heat, which consisted of hot sitz baths two or three times a day and the application of an electric hot pad to the painful areas at night. Of these 28 patients, 25, or 90 per cent, were relieved and only 3, or 10 per cent, were not. The third group was treated by massage and surgical removal of foci of infection. This group consisted of 10 patients. Nine, or 90 per cent, were relieved and 1, or 10 per cent, were unrelieved.

SUMMARY AND CONCLUSION

It is emphasized that a spasm of the levator ani and coccygeus muscles has been observed in patients with coccygodynia or "television bottom." Not infrequently a spasm of the piriformis muscle, with pain in the upper portion of the gluteal region and pain down the back of the thigh, may accompany coccygodynia.

In the series of cases here reported, a reasonably good result was obtained by massaging the spastic muscles and removing the foci of infection when necessary. A determined effort was made to instruct all patients with regard to correct posture. A special effort was made to teach them to lean forward and to sit well back in a chair or car seat. Surgical removal of the coccyx has been condemned as a treatment for coccygodynia.

In a series of 100 patients, 62 were treated only by massage, with 80 per cent relieved of symptoms; 28 by instructions for proper posture and by heat, with relief in 90 per cent. Ten were treated by massage and surgical removal of foci of infection, with relief of symptoms in 9 cases.

ZUSAMMENFASSUNG UND SCHLUSSFOLGERUNG

Es wird hervorgehoben, dass bei Kranken mit Steissbeinschmerz oder "Television-Hintern" ein Krampf des M.levator ani und der Steissbeinmuskeln beobachtet wird. Nicht selten ist die Kokzygodynie mit einem Krampf des M.piriformis mit Schmerz im oberen Abschnitt der Gesässgegend und abwärts entlang der Hinterfläche des Oberschenkels vergesellschaftet.

In der Krankenserie, über die hier berichtet wird, wurde ziemlich gute Erfolge durch Massage der verkrampften Muskeln und, wenn nötig, durch Entfernung von Infektionsherden erzielt. Es wurden zielbewusste Anstrengungen gemacht, alle Kranken über richtige Körperhaltung zu belehren. Besonders bemühte man sich, ihnen beizubringen, sich vorzubeugen und weit hinten im Stuhl oder im Autositz zu sitzen. Die chirurgische Resektion des Steissbeins wird als Behandlung der Kokzygodynie abgelehnt.

Unter 100 Patienten wurden 62 ausschliesslich mit Massage behandelt, und 80 Prozent von ihnen erfuhren eine Befreiung von ihren Beschwerden. 28 Patienten wurden durch Belehrung über richtige Haltung und mit Hitze behandelt, und 90 Prozent von ihnen verloren ihre Beschwerden. Von zehn Patienten, die mit Massage und chirurgischer Entfernung von Infektionsherden behandelt wurden, wurden neun beschwerdefrei.

RÉSUMÉ ET CONCLUSIONS

Un spasme des muscles releveurs de l'anus et ischio-coccygiens a été observé chez des malades atteints de coccygodynie. Il n'est pas rare que cette affection soit accompagnée d'un spasme du muscle pyramidal, avec douleurs dans la partie supérieure de la région fessière et douleurs descendant le long du dos de la cuisse.

Dans la série de cas décrits ici, des résultats assez satisfaisants ont été obtenu par le massage et l'élimination de foyers d'infection lorsque c'était nécessaire. Les malades ont été instruits au sujet de la position correcte à adopter, aussi bien dans la position debout qu'assise. L'ablation chirurgicale du coccyx est rejetée par l'auteur dans les cas de coccygodynie.

Sur 100 patients, 62 ont été traités par massage seulement, avec soulagement des symptômes dans 80% des cas, 28 ont été traités par la chaleur, avec instructions quant à la possition correcte à adopter, avec soulagement dans 90% des cas. 10 cas ont été traités par le massage et l'élimination chirurgicale de foyers d'infection, avec soulagement dans 9 cas.

RIASSUNTO

Nei malati con coccigodinia si è osservata la presenza di uno spasmo dell'elevatore dell'ano e dei muscoli coccigei; talvolta anche il muscolo piriforme è contratto e vi è dolore nella parte alta della regione glutea e nella regione posteriore della coscia.

Nei casi osservati si ottennero dei risultati discreti mediante il massaggio dei muscoli spastici e l'allontamento degli eventuali foci di infezione. Si diede molta importanza all'istruzione dei malati circa la posizione piú corretta da tenere, cioè sedersi ben indietro sulla sedia o sul sedile dell'automobile, stando inclinati in avanti. L'asportazione del coccige è sconsigliata.

Di 100 malati, 62 vennero curati solo con il massaggio, e l'80% di essi ebbe la scomparsa dei disturbi; negli altri 28 si consigliò la posizione adatta e si fecero applicazioni calde, con il 90% di guari-

gioni. Nei 10 rimanenti si fecero massaggi e asportazione chirurgica dei focolai di infezione, con 9 guarigioni.

SUMARIO E CONCLUSOES

Diz que um espasmo do elevador do anus e os musculos coccigeos foi observado em pacientes com coccigodinia ou "botão de televisão." Não é raro registar tambem espasmo do musculo piriforme com dor na porção superior da região glutea e face posterior da coxa. Nos casos aqui apresentados resultados rasoaveis foram obtidos por massagem muscular e remoção de focos infecciosos. Instrue os pacientes sôbre a postura correta. Condena a excisão dococcix, como tratamento da coccigodinia. N'uma serie de 100 doentes 62 foram tratados por massagem com 80% de alivio; 28 por instruções sobre postura e por termotera pia com melhora em 90%. Dez foram tratados por massagem e remoção de focos com alivio de sintomas em 9 casos.

RESUMEN Y CONCLUSIONES

Se llama la atención sobre el espasmo del elevador del ano y de los músculos coxígeos en los enfermos afectos de coxigodinia o "televisión botton" Con frecuencia la coxigodinia puede acompañarse de espasmo del piramidal con dolor en la parte alta de la región glútea y en la cara posterior del muslo.

En una serie de casos presentados se obtuvo un razonablemente buen resultado por el masaje de los músculos espasmodizados y la eliminación de los focos asépticos.

Se debe, además, instruir al enfermo en relación con la importancia de adoptar una postura correcta. Se debe intruir especialmente en la forma de inclinarse hacia adelante y de sentarse bién hacia atrás en las sillas o en el asiendo del coche. La extirpación quirúrgica del coxis debe condenarse como método para curar la coxigodìnia.

En una serie de 100 enfermos 62 fueron tratados solamente por masaje, con un 80% de desaparición de los síntomas; otros 28% fueron tratados posturalmente y con aplicaciones de calor con mejoría en un 90% de 1 los casos.

De 10 enfermos tratados con masaje y extirpación del coxis 9 curaron completamente.

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Medicine is a most difficult art to acquire. All the college can do is to teach the student principles, based on facts in science, and give him good methods of work. These simply start him in the right direction; they do not make him a good practitioner—that is his own affair.

—*Osler*

Pupillary Block in Phakic and Aphakic Eyes

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DETROIT, MICHIGAN

PUPIL block is a phenomenon that occurs in phakic and aphakic eyes only in special circumstances. It has been recognized in aphakic eyes since 1865 (Bowman;[1] Urbanek[2]) and in phakic eyes since the recognition of iris bombé, but has drawn particular attention in eyes with angle-closure glaucoma since 1952 (Chandler;[3] Haas and Scheie[4]).

The special circumstances necessary for pupil block depend on resistance to the outflow of aqueous through the pupillary space in eyes with no functioning extrapupillary pathway for the circulation of aqueous. This may occur in the following circumstances:

Phakic Eyes:

1. Seculusio pupillae, due to iridocyclitis. It may also occur with iridectomy when an inflammatory membrane is present.
2. Spherophakia.
3. Instumescent cataract and after discission of congenital cataract or after traumatic rupture of the anterior capsule.
4. Angle closure glaucoma.
5. Malignant glaucoma.
6. Dislocation of the lens into the anterior chamber.
7. Posterior subluxation of the lens in which the pupil is blocked by relatively "solid" vitreous.
8. Air blockade after cyclodialysis.

Aphakic Eyes:

1. In the presence of aphakic, with or without iridectomy, in which the pupil is blocked by a membrane, usually inflammatory.
2. After extracapsular cataract extraction, when cortical material blocks the pupil and iridectomy openings.
3. After intracapsular cataract extraction. The pupil is blocked by adhesion of the iris to the intact hyaloid membrane.
4. After discission of cataract, usually when no iridectomy is present and the pupillary opening is blocked by the hyaloid, the posterior capsule, an inflammatory membrane or "solid" vitreous.
5. In air blockade after cyclodialysis.
6. In air blockade after cataract extraction.

The mechanism in each of these will be briefly considered, beginning with phakic eyes. In *seclusio pupillae* the passage of aqueous through the pupil is blocked mechanically by adhesions, as is well under-

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Understanding of pupillary block is important to knowledge of the physiologic mechanisms that may lead to increased intraocular pressure in phakic and aphakic eyes under varying circumstances. Five cases are reported.

stood. As new aqueous forms, the pressure behind the iris is greater than in front of it. The iris bulges forward, blocking the chamber angle so that there is a double block to aqueous circulation. If an iris opening is made early by transfixion, iridotomy or iridectomy, before adhesions have taken place between the trabecular wall and the periphery of the iris the aqueous circulation is reestablished and the iris falls back. If, however, adhesions already block the angle, the formation of a hole in the iris frees only one part of the double blockage, so that the iris falls back but the glaucoma continues. This mechanical principle will be considered several times.

The rare disease *spherophakia* is the topic of concern. This is a developmental deformity of the lens, sometimes occurring as part of a hereditary syndrome. The lens is small and spherical. When the pupil is of such size the small lens may bulge into it, the circulation of aqueous through the pupil is blocked and the iris-lens diaphragm is pressed forward, leading to iris bombé and angle block. When the pupil is freed from contact with the lens, the aqueous circulation returns to normal. Treatment consists of the use of mydriatics, with eventual removal of the crystalline lens within its capsule.

The mechanism of pupil block in eyes with *intumescent cataract* is exactly the same as in cases of angle-closure glaucoma, the next condition to be discussed, so it will not be considered separately. A similar situation occurs after simple discission of congenital cataract or after traumatic rupture of the anterior capsule associated with intumescence of the lens substance.

The anatomic basis of *angle-closure glaucoma* is a relative forward position of the crystalline lens and iris in relation to the anterior chamber angle. Pupil block apparently results from the posteriorly-directed vector of force exerted by the iris sphincter when this portion of the iris is bowed forward by the central part of the lens, especially when the pupil is constricted to about midposition. Resistance to the flow of aqueous through the pupil into the anterior chamber results in a higher pressure behind the iris than in front of it. This in turn leads to the so-called physiologic iris bombé, in which the peripheral portion of the iris is forced forward against the trabecular spaces in an eye with an already anatomically narrowed angle entrance, especially true when the pupil is mid-dilated so that the iris can "billow" forward. When the pupil has been contracted to 1 or 2 mm. the iris cannot "billow", and the resistance to deformity of the iris is enough to break the pupillary block. One might picture the iris in these instances as a sail, which is taut when the pupil is small and billowed out by the wind when mid-dilated. The importance of the pupil block phenomenon in these cases is shown by the "curative" effect (when no angle block is present) of peripheral iridectomy, which allows the aqueous to pass directly from the posterior to the anterior chamber, bypassing the pupil. Pupil block, then, does not result in the bombé effect. The choice of broad nasal iridectomy or iridencleisis depends on the need for relief of any concomitant trabecular obstruction.

In the surgical complication called *"malignant glaucoma,"* which occurs most frequently after filtering operations for simple glaucoma, the pupil block results, in my opinion, *I belive,* from luxation of the lens. The lens comes completely forward, forcing the iris against the cornea and blocking both the pupil and all iridectomy openings. Removal of the lens in these circumstances is imperative. It is usually preceded by posterior sclerotomy, which I have found to be of no avail and unnecessary in my own cases. A broad iridectomy should be made at the time of the lens extraction. I have had no expe-

rience with incision of the hyaloid or of the vitreous, which are advised if the lens extraction fails.

Dislocation of the lens into the anterior chamber differs from the forms of pupil block just described in that the border of the pupil is pressed from behind against the posterior surface of the lens. The lens and iris are pressed forward until they touch the cornea and block the trabecular spaces. The lens must be removed early to relieve the glaucoma.

In eyes with *subluxation of the lens* behind the pupil, in which the lens is partly forward and the aphakic part of the pupil is blocked by relatively solid vitreous, a peripheral iridotomy or iridectomy is indicated to prevent the possibility of angle block. When angle block (with iris bombé) has already occurred, both cyclodialysis and peripheral iridectomy are necessary to relieve the pupil and angle block, just as in the cases which will be discussed under aphakic pupil block. Otherwise the lens must be removed.

Air blockage glaucoma was first noted in 1938 by Barkan[5] after the injection of air into the anterior chamber for control of hemorrhage following cyclodialysis. It was noted in both phakic and aphakic eyes. Barkan was able to relieve it by allowing the escape of the air. He realized that the glaucoma was due to obstruction of the free circulation of fluid by pupil block and the subsequent iris bombé effect.

Pupil block in Aphakic Eyes.—In aphakic eyes pupil block may occur without glaucoma, but if allowed to continue untreated it always leads to angle block and secondary glaucoma. It results from a combination of blocking of the pupil opening as well as absence, closure or blocking of all iridectomy or iridotomy openings between the anterior and the posterior chamber. The pupil block itself is due to close contact or adhesion between the hyaloid membrane and the pupillary border, or the presence of an inflammatory membrane secluding the pupil. In some cases there is presumably complete circumferential adhesion of the posterior surface of the iris to the intact hyaloid membrane, even though the pupillary border itself may be completely or partly free.

These requirements for pupil block in aphakia occur usually in round pupil extractions in which the peripheral iridotomy or iridectomy openings are closed by inflammatory membranes or adherence to the surgical wound. Iridotomies certainly can close more easily than iridectomies. Iridectomy or iridotomy openings should not be made in the far periphery or, if they are, should be reasonably large. Two iridectomies are better than a single one. Although prolapse of the iris predisposes to closure of iris openings, prolapse rarely occurs in such cases with good wound closure.

Late shallowness of the anterior chamber without evidence of wound leak or choroidal separation should lead one to suspect the presence of pupil block, even before any elevation of intraocular pressure occurs. It should be particularly suspected when there is any elevation of tension. Chandler[6] used a base tension value of 22 mm. of mercury in designating pupil block, but it is more likely that pupil block can occur even in the presence of hypotony if there is marked suppression of aqueous formation. It is probable that in the postoperative period after cataract extraction such a suppression of aqueous formation may occur.

One may observe an uneven depth of the anterior chamber in some cases, when an inflammatory membrane stretches across part of the chamber and prevents the entire chamber from becoming shallow (Chandler[6]). Such an occurrence should arouse suspicion of pupil block. In 1 of the cases to be described this phenomenon was beautifully demonstrated.

Biomicroscopic study of the anterior segment in a case of pupil block in aphakic eyes after uncomplicated cataract extraction always reveals herniation of the hyaloid-covered vitreous into the anterior chamber. In the child with normal vitreous, after discission, free vitreous probably can produce block in the same manner. In the adult, however, it is unlikely that free vitreous herniating through a rent in the hyaloid membrane produces such block.

The question of mydriasis or miosis as a means of relieving pupil block and vitreous hernia in aphakic eyes has been answered in both ways. Reese[7] advised pupillary dilation with 10 per cent phenylephrine, then constriction with isofluorophate. Kronfeld[8] advised the use of mydriatics. If the pupil is completely free, mydriasis so induced should relieve pupil block. In cases such as those here described, however, it is likely that adhesions between the posterior surface of the iris and the hyaloid membrane, even if incomplete, make either miotics or mydriatics ineffective.

After *discission of cataract,* particularly when no iridectomy is present and the pupil is blocked by vitreous hernia, if the vitreous is relatively "solid," pupil block may occur. The pressure of aqueous behind the iris then produces iris bombé and blocks the chamber angle. If, just before the discission knife is removed, a peripheral iridotomy is made, pupil block may be bypassed and glaucoma prevented.

Air blockade after cyclodialysis in phakic eyes has been discussed. The same may occur in aphakic eyes. Barkan[8] also noted air blockade glaucoma after cataract extraction. In these cases the air must block both the pupil and the coloboma or iridectomies.

The treatment of pupil block in both phakic and aphakic eyes may be summarized as follows: If no increase in intraocular pressure is present (i. e., the angle is still unblocked) iridotomy or, preferably, iridectomy is all that is necessary to bypass the interference with communication between the anterior and the posterior chamber. In doing discission with a needle knife for congenital cataracts, a peripheral iridotomy should be done with the knife just before it is withdrawn from the eye.

In aphakic eyes, if the intraocular pressure is elevated, both the angle block and pupil block must be relieved. A combination of cyclodialysis and iridotomy is therefore essential. In some instances it may be wise to do the iridotomy as a first procedure, to determine whether the angle block is permanent, and then do the cyclodialysis. Gonioscopy is certainly more helpful when the chamber is deepened.

In cases to be presented, pupillary block followed round pupil cataract extraction, with two peripheral iridectomies in each instance. In each case three to five corneoscleral sutures, with a limbal-based flap, were used. The sutures included one preplaced McLean suture and two to four postplaced sutures of the same type. The possibility of wound leak associated with loss of the anterior chamber was considered because of late emptying of the anterior chamber, but was not observed. No choroidal separation could be detected. Each case differed in its course, yet all had certain basic similarities. In each the hyaloid membrane remained intact. In each both peripheral iridectomies became blocked, although this was not recognized at first. In each instance the pupil could be dilated and constricted to some extent. In each the anterior chamber formed normally after the operation but became shallow only after the early postoperative period, and in each it became deep after successful drainage of aqueous humor plus reestablishment of flow between the posterior and the anterior chamber. In 1 case treatment

was possible before the angle became blocked and thus before glaucoma had developed.

REPORT OF CASES

CASE 1.—Mrs. M. B., aged 60, underwent an operation for extraction of an immature cataract on July 8, 1954. A McLean flap and three McLean sutures were used. An attempt at intracapsular extraction was unsuccessful, and the capsule broke. The posterior cortex remained after the nucleus and most of the capsule had been removed. Two peripheral iridectomies were made. About two weeks after the operation the cortical material swelled and filled the pupillary space, the chamber became shallow, after which, on July 31, the tension rose to 48 mm. Schiotz. The chamber remained shallow. August 19 a peripheral corneal incision was made 1.5 mm. from the upper limbus, and the lens material was irrigated out. A reverse cyclodialysis was made in the upper temporal quadrant, but the anterior chamber remained shallow and the tension became elevated, slightly at first, then up to 60 mm. Hg. (Schiotz). On October 1 a cyclodialysis was done through a scleral incision 4 mm. from the limbus in the lower temporal quadrant. At the same time a peripheral iridectomy was made in the upper nasal quadrant, and air was injected into the anterior chamber before closure. Recovery was uneventful. The tension and corrected vision were normal up to the last most recent examination, in December 1957.

In this case it was noticed during convalescence that the swelling of the remaining posterior cortical material occluded the pupil. It was not realized that the iridectomies were obstructed until after cyclodialysis failed to deepen the anterior chamber, in spite of the fact that cortical material had been washed out and the pupil no longer appeared obstructed. An iridectomy-cyclodialysis permitted drainage through the obstructed angle and between the anterior and posterior chambers. The chamber deepened thereafter.

CASE 2.—Mrs. B. B., aged 63, had undergone round pupil intracapsular cataract extraction on Dec. 12, 1957. A McLean flap and five corneoscleral sutures were used. Two iridectomies had been made in the far periphery of the iris. Recovery was uneventful. On Jan. 28, 1958, anterior chamber was noted to be shallow, with the hyaloid membrane bulging forward. The pupil border was adherent to the hyaloid membrane in places but was generally free. No choroidal separation or wound leak was present. The tension was 40 mm Hg. (Schiotz 1955 scale). Atropine failed to improve the condition. Phospholine iodide similarly was ineffective. The chamber angle was blocked, and the locations of the iridectomies could barely be seen in the periphery. A cyclodialysis was done on February 19, with local anesthesia, in the inferior temporal quadrant, but, although the operation was uncomplicated, the tension remained elevated. On March 20 a cyclodialysis was done at the 12 o'clock area, just at the level of the insertion of the superior rectus muscle. Air was injected, and a Wheeler knife iridotomy was made in the temporal iris. The patient was seated on the operating table and was taken back to her room in a wheelchair. The anterior chamber was deep the next day and has remained so. The tension has been soft. An open cyclodialysis cleft is present.

This case is important in showing the lack of value of cyclodialysis unless it produces a communication between the anterior and posterior chambers. When such a communication was established, the anterior chamber deepened and the vitreous bulge receded.

CASE 3.—U. G. N., aged 50, underwent a right round pupil intracapsular cataract extraction with two peripheral iridectomies on April 24, 1957. A McLean flap and five corneoscleral sutures were used. Convalescence was uneventful, and on June 11 the corrected vision was 20/20 in this eye. The anterior chamber was of normal depth. On October 3, shallowness of the anterior chamber was noted. The tension was 17 mm. (Schiotz 1955 scale). No wound leak or choroidal separation was present. Bulging of the intact hyaloid membrane, nearly touching the cornea, was evident. The pupil border was free. The angle was shallow but open. The peripheral iridectomies were closed. Mydriasis and miosis had no effect on the depth of the anterior chamber. On October 19 a Wheeler knife incision was made temporally through the cornea and peripheral iris. On the following day the anterior chamber was deep. The tension remained normal. In August 1959, however, the iridotomy wound closed and the chamber became shallow. Presumably the pigment epithelium of the iris may proliferate to close such an opening. A periph-

eral iridectomy solved the chamber depth problem.

This case is the first described in which the late development of a shallow anterior chamber led to the relief of pupil block before angle block occurred. It also demonstrates that a small iridotomy may close and thus is less effective than a peripheral iridectomy.

CASE 4.—T. H., a 60-year-old man underwent a left intracapsular cataract extraction on Nov. 5, 1958. Two peripheral iridectomies were made. The postoperative course was entirely uneventful until, on the ninth day, the severe pain and redness of the eye developed. When he was seen, fibrin was present in the pupillary area. A diagnosis of acute purulent endophthalmitis was made, and treatment was started with an antibiotic administered locally and orally. Atropine and steroids were also employed. The eye responded well, and a fine pupillary membrane formed. The peripheral iridectomies closed. The nasal portion of the iris ballooned forward as in iris bombé. The tension was 29 mm. (Schiotz 1955 scale). The angle was one-third open; pupil block was diagnosed. On January 22 a cyclodialysis was done, followed by an iridotomy done temporally with a Ziegler knife. The eye healed uneventfully, and the localized iris bombé became flat. The tension remained normal and the eye quiet. Vision is reduced to 20/400 because of marked vitreous opacities.

CASE 5.—R. L., a 67-year-old man, underwent a left cataract extraction on Feb. 4, 1959. Two peripheral iridectomies were made, and the lens was removed, a Bell erisiphake being used. The capsule ruptured, but the impression was that most of it had been removed. On the following day, however, it was possible to see some cortical material in the pupillary area. The course was uneventful until the fourth postoperative day, when the patient suddenly began to have severe pain. It was my opinion that the cortical material had caused pupillary block by obstructing the pupil and iridectomies. The patient was taken to the operating room, where the eye was observed to be hard. The central corneoscleral suture was removed and the wound opened sufficiently to grasp the iris and complete the iridectomy. Some cortical material was irrigated out. A new corneoscleral suture was inserted and atropine instilled. The course was uneventful. The corrected visual acuity is 20/20.

SCHLUSSFOLGERUNGEN

Kenntnis der Pupillensperre ist wichtig zum Verständnis der physiologischen Mechanismen, die unter verschiedenen Umständen bei Augen mit Linse oder bei linsenlosen Augen zur Erhöhung des intraokulären Druckes führen können.

CONCLUSIONS

Il est important de connaître le mécanisme du blocage pupillaire pour comprendre les mécanismes physiologiques pouvant provoquer une augmentation de la pression intra-oculaire dans les cas de phakitis et d'aphakie.

CONCLUSIONI

Il riconoscimento del blocco pupillare é importante per la comprensione dei meccanismi fisiologici che possono condurre all-aumento della pressione intra-oculare nei vari casi.

CONCLUSIONES

El reconocimiento del shock pupilar es importante para comprender los mecanismos fisiológicos que pueden conducir al aumento de la presión intraocular en los ojos fáquicos y afáquicos en diversas circunstancias.

CONCLUSOES

O conhecimento do bloqueio pupilar é importante na compreensão dos mecanismos fisiologicos que promovem uma hipertensão intraocular em diversas circunstancias.

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The Weight Stream in Charcot's Disease of the Joints

Reconstruction of the Charcot Knee

MICHAEL BURMAN, M.D.

NEW YORK CITY, NEW YORK

CHARCOT'S disease is the sequel of the weight force striking an insensitive joint. Joint sensation resides in the ligaments and in the synovial membrane. The *insensitive and avascular cartilage* is protected from undue compression and shear by a proprioceptive joint sense, reflexly governed. The widespread reflex loss which deprives a joint of proprioceptive sense results in deafferentation.

The resultant weight force (W) passes through the center of the knee or a little medial to it and more posteriorly than anteriorly. It strikes the convexity of the medial femoral condyle and then the concavity of the medial tibial condyle. The differential elements of the weight force diverge as they strike the tibial concavity. The shear component of the weight force, whose angle cannot exceed 45 degrees (Poorman) disintegrates the medial tibial condyle. A vicious circle is set up. The downward and medial tilt of the medial tibial condyle causes bowing of the knee, the typical lateral deformity of a Charcot knee. The weight force acting on this bowed knee gives more destruction of the tibial condyle, as well as more tilt and more bowing. With maximal shear the femur may slide down the tibia. This lateral luxation of the leg may rub the lateral femoral condyle into an oblique line, an event demonstrated by Steindler. Very uncommonly, the medial femoral condyle is sliced posteriorly and is then displaced by pull of the medial head of the gastrocnemius.

The weight-ordered destruction of the

The Charcot joint is made insensitive by the loss of proprioception. The destruction of the medial tibial condyle follows as the effect of the shear component of the weight force, whose maximal angle is 45 degrees.

Reconstruction of the joint should break up the vicious circle of destruction that results in the vicious circle of deformity, further destruction and further deformity.

Reconstruction does not give the full security of fusion, but it stays the disease if infection does not follow the operation. It should be done early rather than late when retention of motion is desired, or in one knee if there is neuropathic disease in both.

The use of the patients own patella is probably better than a foreign substance, such as the metal or plastic mold, which the neuropathic joint does not seem to tolerate.

From the Hospital for Joint Diseases, New York.
Submitted for publication July 30, 1959.

medial tibial condyle is not *specific* of neuropathic change in the knee joint. I have seen it in cases of rheumatoid arthritis and of osteoarthritis of the knee joint. (A Charcot joint reacts *excessively* in the hypertrophic form of the illness. The effect of shear is *maximal* both in the atrophic and the hypertrophic form of neuropathic joint disease.)

I had not thought valgus deformity possible in Charcot's disease of the knee, but it exists, following lateral displacement of the weight force. It is to be differentiated from osteoarthritis of the knee with great destruction of the lateral tibial condyle. I saw an excellent specimen (No. S87-12) of a Charcot knee in valgus in the Museum of the Royal College of Surgeons in London.

Figure 1, *A* and *B*, shows the effect of weight on the medial femoral condyle and the medial tibial condyle, the initial greater

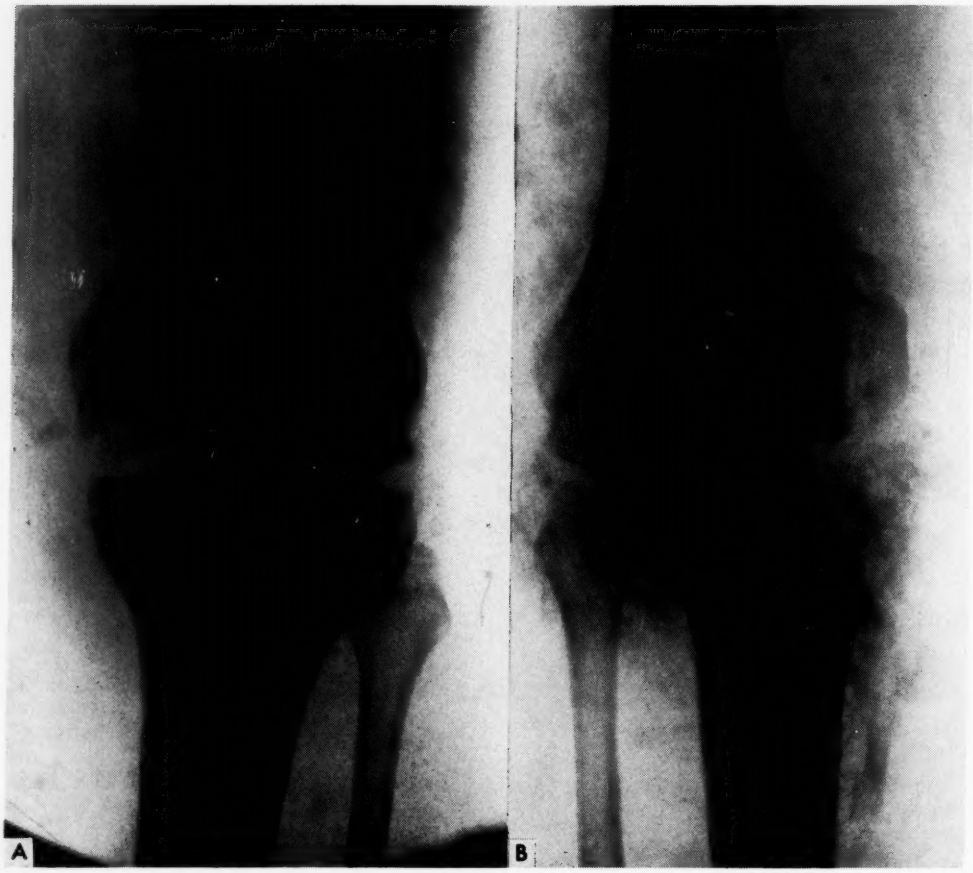


Fig. 1.—*A*, the effect of weight on the medial tibial condyle. The left knee of a colored male adult. Anteroposterior roentgenogram of Nov. 13, 1940. The medial joint space is wider then the outer. The medial tibial condyle seems intact. The medial femoral condyle is fragmented. *B*, anteroposterior and lateral roentgenograms taken June 20, 1944. There has been great advance of Charcot's disease. Medial tibial condyle is extensively destroyed, much more so than corresponding femoral condyle (print reversed).

effect being on the medial femoral condyle. The advance of the illness destroyed the medial tibial condyle more than the medial femoral condyle. Figure 2 depicts the end event of uncontrolled disease, with medial and downward luxation of the thigh on the leg. The disease may be stemmed by fibrosis and new bone formation, but this is often not successful.

There is no operation that can cure Charcot's disease of a joint. The surgeon is between the Scylla of resection and the Charybdis of fusion. Resection of a small joint, such as the metatarsophalangeal joint of the great toe, can stay the illness. The end effect of illness in a large joint, such as hip, knee or ankle, is the literal self-resection of the joint and consequent instability deformity. The "broomstick" of a Charcot hip may be angulated in the Schanz manner and the joint made stable.

Joint resection is a poor solution to the problem of the Charcot knee, and the attempt is made to stiffen the knee by brace, by fibrosis or by bony fusion. Midthigh amputation is the final way out of the dilemma.

The Possible Operative Procedures. — Synovectomy and "Housecleaning": Synovectomy does not necessarily stay the progress of Charcot's disease. It does not restore proprioceptive sense to the joint.

I have observed at least 3 cases in which synovectomy was done. In 1 the surgeon did the synovectomy three months after the injury, which occurred on Feb. 13, 1936, and before the neuropathic nature of the joint affliction was clear. It became quite evident two months later.

Amputation: An above-the-knee-amputation is sometimes needed if the Charcot joint becomes infected or if, in the presence of bilateral neuropathic disease of the knees, one joint is destroyed to such an extent that it cannot be supported properly, there is adverse weight transfer to the opposite joint.

Fig. 2.—Negress aged 68 with Charcot's disease of right great toe and left knee. This roentgenogram, taken on Oct. 11, 1945, is distinct enough to show end stage of destruction of medial tibial condyle. Tibial joint surface becomes oblique, and femur luxates medially on this inclined surface. This is a good example of shear, a vicious slicing force which becomes increasingly greater as joint disintegrates. Greatest angle of shear is 45 degrees.

CASE 1.—A 45-year-old Negro underwent a fusion operation on a left Charcot knee on Aug. 25, 1930. The joint surfaces were denuded of cartilage and the patella removed. A sliding bone graft was passed between the tibia and the femur anteriorly and countersunk. Bits of the patella were used as a bone pack. Many postoperative notes stated that the joint was *stiff* and that the patient could walk as much as he wanted to.

The right knee, later the site of a recurrent painful effusion, became unstable. Synovectomy was done on Aug. 13, 1935. The joint

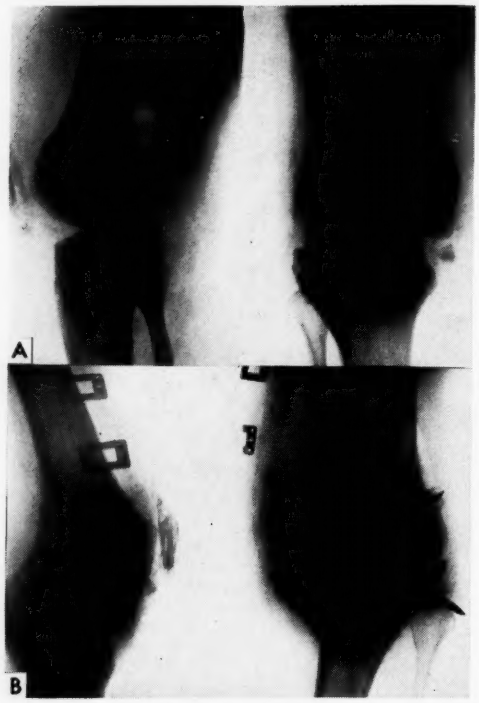


Fig. 3.—*A*, left Charcot knee of a 59-year-old man. Note unusual fragmentation of the patella. Fusion of knee was done on Oct. 27, 1942, by apposition of raw bony surfaces of femur and tibia and nailing in X manner. Sclerotic juxta-articular bone was drilled at many points. Disintegrated patella was removed. *B*, failure of fusion with increasing disintegration of the internal tibial condyle. Varus of knee forced point of nail through skin in region of fibular head. Nail was removed on July 31, 1943. Patient died in September of subarachnoid hemorrhage and syphilitic endarteritis of brain.

continued to disintegrate, however, making above-the-knee-amputation necessary on Oct. 7, 1936.

A soft tissue abscess formed on the anteromedial part of the *left* knee and was drained on Feb. 15, 1941. The joint now had about 5 degrees of motion, and the roentgenograms showed a joint line. The patient wore a brace and used crutches. The knee was a little painful on April 11, 1946.

Synovectomy failed to halt Charcot's disease of the right knee. The fusion of the left knee was fibrous and not bony, since the roentgenograms failed to show through-and-through trabeculation. A thin joint line persisted. The great strain thrust upon this knee after amputation of the other leg broke the previously adequate fibrous fusion.

Fusion of the Knee: This operation must be done before disintegration of the joint prohibits any intervention except amputation. There are many failures and few successes. Vishoot stated that one-half of the attempted operations fail. Long periods of postoperative immobilization are often demanded. These statements were only too well confirmed by the various discussants at the 1957 meeting of the Australian Orthopedic Association. One of them, Keon-Cohen, even asked why a Charcot knee should be fused. Horwitz noted that the results of fusion of a neuropathic joint have been uniformly poor and that even in the knee joint, which is best suited for operation, the achievement of successful fusion merits report.

Disturbed proprioception must persist in the raw bony surfaces. Sclerotic bone, even if drilled, does not favor fusion. If intra-articular fusion is to succeed, there must be firm coaptation of broad cancellous bone surfaces under reasonable compression. There are many technics of coaptation and compression that employ intramedullary or extramedullary fixation.

I need not review the literature. These figures probably tell the story a more optimistic story than the full truth, for there must be many who have not recorded their failures.

Time alone tells whether fusion is fibrous or bony. At least two years should elapse after operation before the estimate of fusion is certain. The persistence of a joint line denies through-and-through trabeculation. Most authors allow shorter postoperative periods:

Pomeranz and Rothberg: Of 8 fusion operations, 7 failed.

Cleveland and Smith: Of 3 fusion operations, 1 failed.

Cleveland: Of 4 additional knee fusions, 2 failed.

Soto-Hall: 3 successful fusions.

McCauley: Of 6 fusion operations, 1 failed.

Steindler: Of 6 fusion operations, 1 failed, 1 was followed by fibrous union.

Horwitz: Of 12 fusion operations, 10 failed. (Horwitz may have considered the same cases as Pomeranz and Rothberg, since both studies came from the Hospital for Joint Diseases.)

H. Barry: Barry reported that he knew of 1 good result.

A. Watts: This author had 2 cases of *sound* fusion by arthrodesis.

W. Gayton: Gayton succeeded in 2 or 3 attempts to fuse neuropathic joints.

D. Parker: Success was achieved in 1 of 2 cases.

Charnley had 1 success and 2 failures by the use of his compression technic. In his opinion, aside from mechanical failure, the dense sclerotic bone of the Charcot knee predicated failure. His success was obtained in a case in which the bone substance seemed normal. Guevara, who used Charnley's technic, succeeded once and failed once. Newman reported 4 cases in which the Charnley technic plus an intramedullary tibial graft was used. He was convinced that he had obtained fusion, but his longest follow-up period was only seven months.

Morris and Mosiman compared 12 cases of compression fusion with 24 cases of noncompression fusion. Solid ankylosis was achieved in every case but 1. No specific statements were made on fusion of the Charcot knee. In 1 case in the compression group and 2 in the noncompression group fusion was obtained. Roentgen evidence of fusion required six months or more.

Stack and Lichtman, who derived their technic from Chapchal, used an intramedullary nail in 7 cases. Only in their Case 1, followed for eleven months, was the

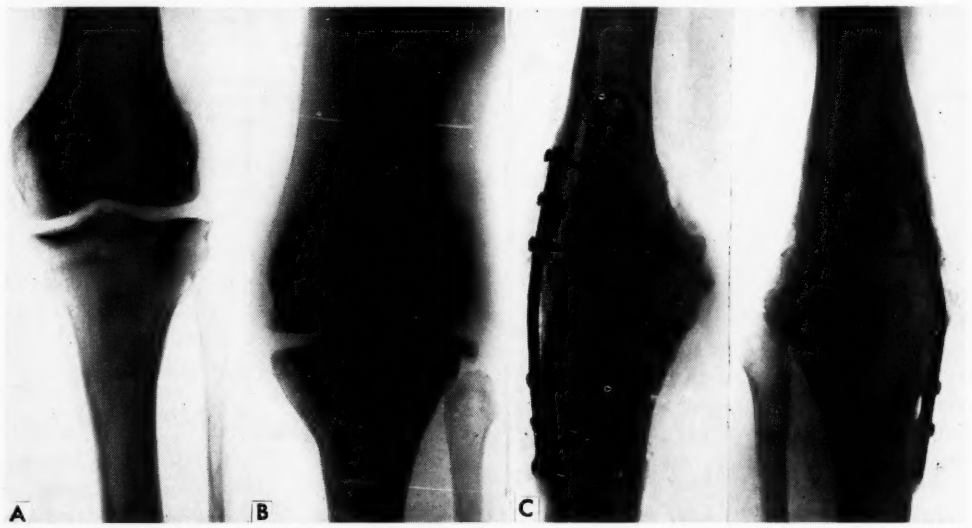


Fig. 4.—*A*, roentgenogram taken Oct. 6, 1944. Knee joint is radiographically normal, aside from spiking of lateral tibial condyle. *B*, roentgenograms taken April 3, 1945, demonstrating destruction of lateral tibial condyle. *C*, post-operative roentgenograms taken Feb. 17, 1956, more than ten and one-half years later. Solid fusion of knee. Note that fusion was done early, before there was too much destruction. (*I am indebted to Dr. Abraham Rothberg for these roentgenogams.*)

knee solid clinically. Amputation was done in another case less than a year after nailing.

Brashear, who also tried this unsatisfactory form of nailing in 3 cases of Charcot's disease of the knee, reported that in 2 of his cases the femur broke at the point of introduction of the nail. In the third case the nail broke at the joint line.

Deyerle fused the Charcot knee of a man 47 years old, using a medullary nail as well as a plaster cylinder. The cast was worn for six months, and the man used crutches all this time except for two weeks. The joint was solid at six months, and he worked daily at heavy labor. The postoperative roentgenograms still showed a joint line, and the fusion was fibrous. Tran-Ngoc-Ninh likewise achieved a stable knee by this technic of nailing, but again with fibrous union.

One intramedullary nail does not give adequate fixation. Possibly two nails should be used.

Ego Grossi (1954) compressed the raw surfaces of the resected knee with two threaded wires, crossed in the manner of an X, nuts and bolts of the Merle d'Aubigné type adjusting the tension within the wires to give stimulating compression of bone. He used the operation in 22 cases with satisfactory results; apparently there was no case of Charcot's disease in this group. Bosworth (1946) used a Smith-Petersen three-flanged nail in fusion of the knee joint. Three Charcot knee joints were arthrodesed, and in 2 the joint became solid. The follow-up period, however, did not exceed a year. In 1 of these 2 cases a previous attempt at fusion had failed. The apposition of raw, broad, bony surfaces made secure against rotation by the nail and the absence of full weight-bearing for about a year were emphasized

Two sets of roentgenograms of Charcot knee joints in which operation for fusion was done are shown. In 1 case failure resulted, for the bone was sclerotic and the crossed nail fixation possibly inadequate (Fig. 3, *A*, *B* and *C*). Figure 4, *A*, *B* and *C*, shows the solid fusion of a Charcot knee of a man who was 62 years old in 1956. The operation was done by Dr. A. Rothberg and myself on May 21, 1945, and the follow-up period is more than ten and one-half years. (I am indebted to Dr. Rothberg for permission to reproduce these roentgenograms.)

Reconstruction of the Knee Joint: I believed and still believe that further deafferentation destruction of the Charcot knee joint could be prevented by horizontal leveling to resist the disintegrative effect of the weight force. Operation might follow one of two plans: (1) removal of the condyle and its replacement by a nonirritating mold made either of a plastic material, such as lucite, or a metal, such as vitallium, or (2) building up of the depressed tibial condyle by means of the patella. The reconstructed joint is neuropathic. Both plans have been tried, a vitallium mold for the replacement of the medial tibial condyle and the autogenous patella for the lateral tibial condyle.

1. *Reconstruction of the Medial Tibial Condyle by a Vitallium Mold.* An indestructible material is substituted for the crumbling cartilage and bone to resist the weight force. The bone receiving and holding a plastic or metal appliance may fail structurally by lack of proprioceptive sense, or disintegration of the femoral condyle may take place as a result of its impact against the mold on motion or on weight-bearing.

The joint was made mechanically sound horizontally to receive the weight force perpendicularly (by overcoming varus). Impact damage of the medial femoral condyle seemed less likely in a joint mechanically restored than in a joint not so restored. The line of weight-bearing became

Fig. 5.—*A*, preoperative roentgenograms taken Aug. 12, 1944. Medial tibial condyle is cupped and fragmented. *B*, vitallium mold, which has shape and size of adult medial tibial condyle. (Made by the Austenal Laboratories.) *C* and *D*, joint at operation on Aug. 12, 1944. Medial tibial condyle was resected. Mold was fixed to tibia by long horizontally placed screw. Little flange was also screwed to bone. Mold was set at a little higher level than the outer tibial condyle, to overcome varus. (*D* and *E*, were taken, originally in color, by Dr. Federico Sotelo-Ortiz. The remaining photographs were taken by Dr. Saul Firtel at the same time.) *E*, resected condyle. *F*, anteroposterior and lateral roentgenograms taken Aug. 15, 1944, showing mold in place.

the normal line of weight-bearing, and the vicious circle of shear was broken.

I have found only one reference to the use of a plastic prosthesis in the treatment of Charcot's disease. One of the 8 neuropathic knee joints reported by Tran-Ngoc-Ninh, the left knee of a woman 52 years old, was resected, with good consolidation in the follow-up period of almost five years. The right knee was held in valgus by destruction of the lateral femoral condyle. The pedicled patella, screwed to the femur

by one long transverse screw, replaced the condyle, but the operation failed owing to nonunion of the patella with the femur. In May 1953 an acrylic prosthesis was used as a substitute for the condyle and held to it by two bolts. The prosthesis shifted when the lower bolt broke. The operation was reckoned a failure.

I used the vitallium mold operation twice. It was perhaps unfortunate that a large metal mold was first tried on the neuropathic joint. Failure of the operative plan (by infection) stopped the use of mold replacement elsewhere. The use of a metal or plastic femoral head often with an endomedullary stem, is now quite popular. More immediate is the fact that *in patients with other pathologic conditions*—fracture deformity, arthritis, or tumor—mold replacement of the femoral condyles of the knee joint (Aufranc and Jones) and of the tibial condyles (MacIntosh) has sometimes been done with success.

CASE 2 (Fig. 5, *A* to *E*).—A mold was placed in the neuropathic right knee joint of a 53-year-old tabetic man on Aug. 14, 1944. The medial tibial condyle was resected. Its articular surface was depressed and saucer-shaped. The cartilage had been "rubbed off." The maximal postoperative temperature was 103.4 F two days after the operation; it was normal on August 24, when he went home, although the plaster had a slight odor. Incision and drainage of the lower half of the wound was done on August 30, when he was readmitted with a high fever. The wound now discharged pus. The organisms isolated by culture were Staphylococcus aureus and a micrococcus overgrowing a streptococcus. The mold was painlessly removed in the patient's bed in the ward on October 10.

Foamy pus continued to be discharged through a 1-inch (2.5 cm.) opening in the lower part of the wound. An extensive operation for incision and drainage was done on Jan. 17, 1945. Since suppuration of the joint persisted, (the contaminating organism being Bacillus subtilis), midthigh amputation was done on March 5. The patient was given an artificial limb on September 6. Kahn and Kline tests of the blood still showed a 4 plus reaction even as late as July 13, 1948. The patient was hospitalized at the Rockland State Hospital from July 7, 1948 to May 22, 1949, for a psychosis with syphilis of the central nervous system.

CASE 3.—Synovectomy and meniscectomy of the *left* Charcot knee of a 50-year-old man were done on March 5, 1943 (a 'housecleaning operation') to relieve pain and recurrent effusion within the joint. The synovium was thick and bits of cartilage and bone detritus were observed in its substance. The result of the operation was good.

The *right* knee became neuropathic a year later, after a fall. The mold operation was done on July 23, 1945. The postoperative course was good. The wound was healed on August 13 except for one point in its lower part. A probe passed through this sinus struck the metal. The synovial fistula became infected by a gram-negative bacillus, a member of the alkaligenese group.

The still securely fixed mold was removed on November 11, fifteen and one-half months after the operation because of persistent pain and a slight discharge through the persisting sinus. The tibia under the mold was lined with a shiny membrane.

On his last visit on July 10, 1947, the patient walked with two crutches and could not bear weight on the right knee. The knee extended to 175 degrees and flexed to 160 degrees. It was in varus of 15 degrees and allowed 10 degrees of lateral motion. The sinus was still open and discharged a foul-smelling fluid.

Roentgenograms show the joint before the installation of the mold, the mold in place and the joint with the mold removed (Fig. 6, *A* to *E* inclusive).

The mold operation failed in both cases. Infection made it difficult to assess the merits or demerits of the mold alone. (These operations were done when antibiotic medication was not available. These experiences confirm the easy susceptibility of the Charcot joint to pyogenic infection and the difficulty of controlling the infection. Steindler reported that 8 of 10 infected Charcot joints needed amputation.)

The adequate postoperative observation of the second case indicated mechanical

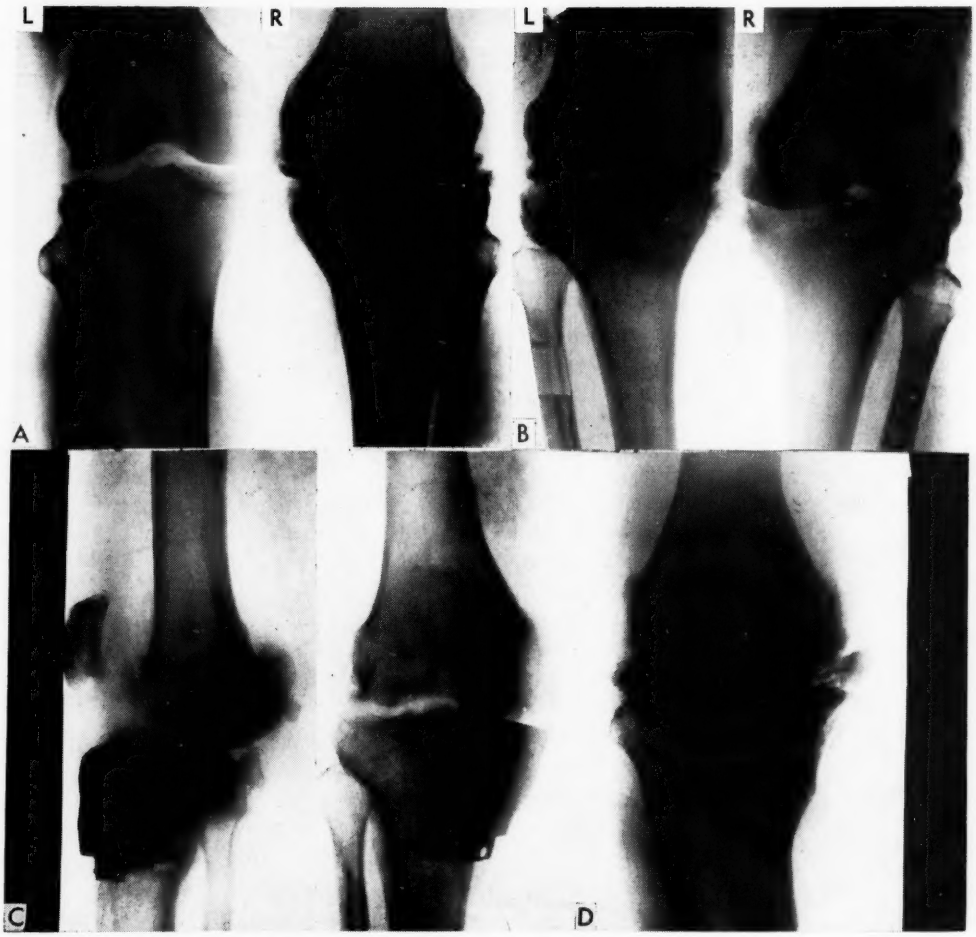


Fig. 6.—*A*, anteroposterior roentgenograms taken Nov. 30, 1943. Right knee *(R)* is normal. Left knee *(L)*, which seemed osteoarthritic, underwent "house-cleaning" operation on March 5, 1943. It is unquestionably neuropathic. *B*, roentgenograms of both knees (May 8, 1945). Both knees are now neuropathic. *C*, vitallium mold in right knee after resection of medial tibial condyle on July 23, 1945 (roentgenograms taken Oct. 30). In comparative roentgenograms of Feb. 26, 1946, mold has not shifted. There was little damage in appearance of medial femoral condyle. *D*, mold removed fifteen months later, on Nov. 11, 1946. (Roentgenogram taken April 17, 1947). Note new bone deposition at site of condylar resection and advance of Charcot disease.

stability of the joint. There was no destruction of the medial femoral condyle by the mold, yet the mold seemed to be too large to be used in a joint as superficial as the knee. A synovial fistula persisted, which became secondarily infected. Larger molds have been used in deeply placed joints, such as the hip, and the wound healed (Moore and Bohlman). There have been recent reports of the use of a hinged metal knee joint (Moeys, Shiers) or an acrylic knee joint (Walldius, Kraft and Levinthal) for conditions other than Charcot's disease. The wounds healed well, and

the metal or plastic material was well tolerated. It must be remembered that none were Charcot joints.

2. *Reconstruction of the Lateral Tibial Condyle in the Very Severe Valgus Deformity of the Charcot Knee by the Use of the Whole Patella.* Failure has a humbling effect, but its lesson may spell later success if the theory of action is sound.

More recently I used the patella to build up the depressed lateral tibial condyle of a Charcot joint in valgus with valgus instability. I had intended to use the vitallium mold, but this could not be done, since the parts, the mold and lateral tibial condyle, were like mirror images. A synostosis of the head of the fibula to the tibia likewise did not permit the use of the mold without resection of the tibiofibular joint. The use of the patella was suggested by Dr. Paul Kotani, a Fellow at the Hospital for Joint Diseases, who was assisting in the operation. I remembered the paper of Wilson and Jacobs and decided to use it.

The use of the autogenous patella had the clear advantage of restoring in good part mechanical stability to the joint like the metal mold. Yet I knew that many things could happen to the graft. It was a free graft of bone, not pedicled as in the case described by Tran-Ngoc-Ninh. It could die. The patella might be countersunk into the tibia by the weight force. It might join the underlying tibia in bony or fibrous union or fail to unite with it. It might move laterally from its position about the curve of the lateral femoral condyle. Fracture, or neuropathic disintegration of the patella, or infection might occur, all to set aside the sought-for stability of the joint.

CASE 4.—On Dec. 1, 1955, through a long median parapatellar incision, the greatly depressed lateral tibial condyle of the right Charcot knee of a man 56 years old was made fairly level with the medial tibial condyle by the use of the whole patella as a graft after "housecleaning" of the joint. (The man had refused arthrodesis.) The cartilaginous face of the patella engaged the lateral femoral condyle. The bony face of the patella was *not* resected to raw bone, nor was it drilled. Bone was packed under the patella, which rested on the raw bony surface of the lateral tibial plateau. The wound healed well, and the knee, first immobilized in plaster, was later supported by an ischial seat, hinged knee-joint caliper brace with which the patient began walking about six weeks after the operation.

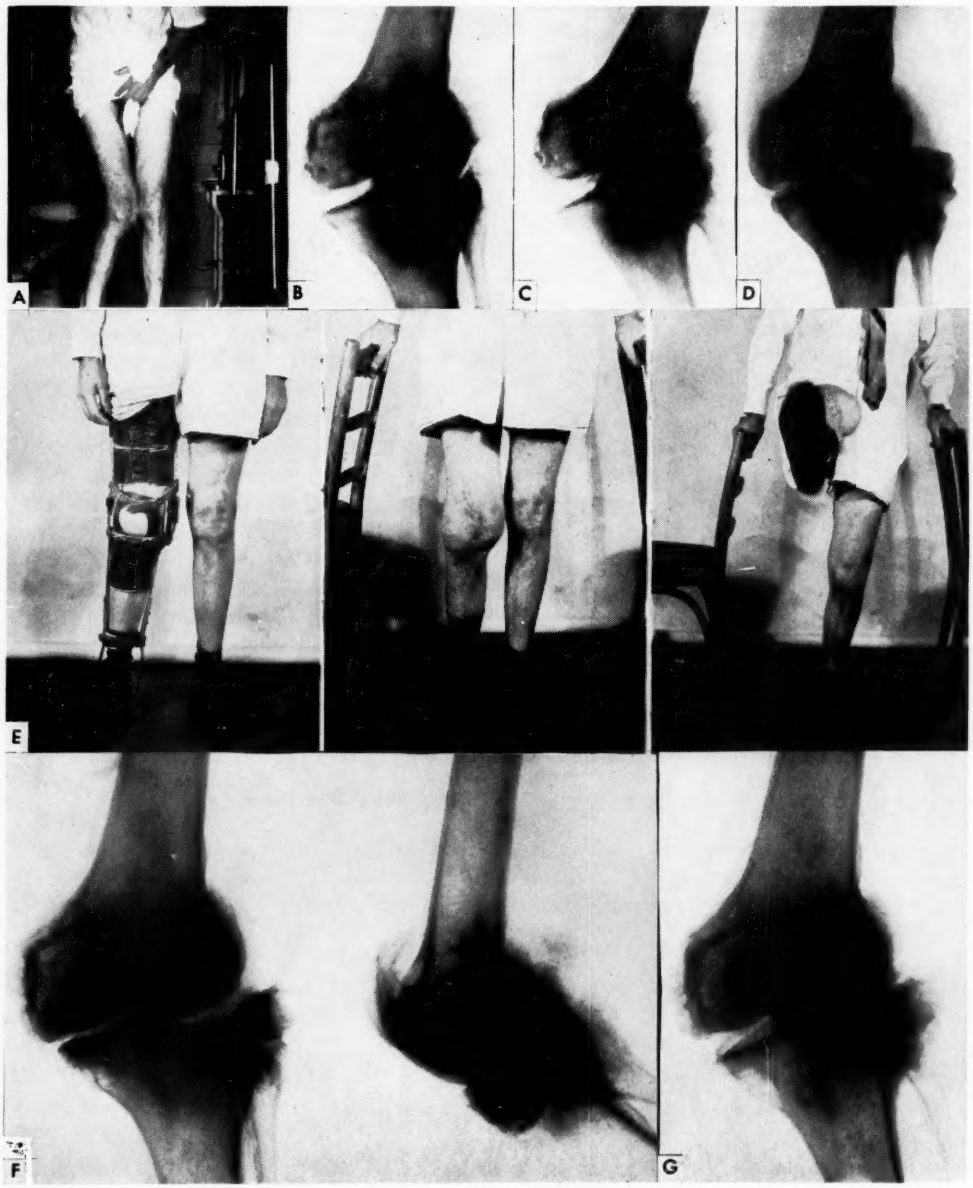
On May 31, 1956, the internal lateral ligament was made tight in order to correct a residual valgity of 12 degrees. The Milch technic was used. The joint was firm after reenforcement of the ligament with a fascial strip and imbrication of the new ligament horizontally and vertically by mattress sutures.

The joint both before and after this operation remained warm and swollen with fluid. Repeated aspirations yielded 20 to 100 cc. of yellow fluid, which became sanguineous at the end of the aspiration. In July the patient became feverish and the joint warmer by pyarthrosis within the joint after an aspiration. The patient was again hospitalized and recovered quickly under antibiotic medication,

Fig. 7 *(opposite).*—*A*, preoperative photograph, originally in color, showing great right knock-knee. *B*, preoperative roentgenograms taken on June 29, 1955, of right knee which is held in valgus by destruction of lateral tibial condyle. There is fusion of superior tibiofibular joint. *C*, a stress film indicating increase in valgus by abduction (June 29, 1955). *D*, anteroposterior roentgenogram taken Feb. 20, 1956, showing patellar implant, which is a brick leveling the joint line. *E*, postoperative photographs, taken Feb. 29, 1956, showing patient standing with brace on, brace off and kicking. Joint contained 40 cc. of serosanguinous fluid. *F*, anteroposterior and lateral roentgenograms of right knee taken on March 7, 1958, showing dense patella over synostotic tibiofibular joint. (Significant roentgenographic observation is rarefaction of tibia at insertion of the medial collateral ligament, an event noted first in roentgenograms of May 1957. Is this a neuropathic tension osteopathy of bone such as is seen in Charcot's disease of bone?) *G*, anteroposterior stress film, in abduction, of extended knee, taken on same day, shows some opening of medial joint space. Compare this opening with that of Fig. 7*C*. Zone of tibial rarefaction is clearly visible. *(B* and *C* are reproduced by courtesy of the *American Journal of Roentgenology, Radium Therapy and Nuclear Medicine.)*

traction, application of an ice bag to the knee and aspiration of the joint. The aspirate was always sterile.

This patient has been under my observation now for three and one-half years. The joint is now cold and no longer holds an effusion. The implant remains firm. He walks fairly well with brace and cane support. Without the brace he walks poorly, because of overextension of the knee. He can extend the knee joint



actively almost to 180 degrees, and in this position there is lateral stability. He can bend it to 80 degrees. There is still some valgus of the knee, but far less than before the operation (Fig. 7, *A* to *G* inclusive).

The operation was reasonably successful, for it achieved two major things: the advance of the Charcot disease in the right knee has been stayed and the vicious circle of destruction and deformity broken. The patella is not a perfect fit, for there is overextension of the knee. The other weight-bearing joints, especially the left knee, have shown no sign of Charcot disease, for they have been spared overweight by adverse transfer of the weight force. The Charcot lumbar portion of the spine has fused naturally.

CASE 5 (Fig. 8).—At my suggestion, Dr. O. C. Kestler did the same operation on Feb. 2, 1956, through a lateral parapatellar incision on the left Charcot knee joint of a 50-year-old woman. The joint was held in valgus. The right Charcot knee was held in the more typical varus deformity. Drill holes were passed through the bone of the lateral tibial condyle after removal of its cartilage. Similar drill holes were made in the anterior fibrous face of the patella. Bone chips taken from the distal end of the femur were wedged between the patella and its tibial condyle. The lateral instability of 45 to 50 degrees was reduced to 10 degrees. The wound healed well.

On April 10 the patient walked with two crutches and could bear weight firmly on the left knee, which was supported by a laced canvas corset. The joint was a little warm but held no effusion. It moved actively between 170 and 90 degrees. There was valgus of about 8 degrees, which could be passively increased a little. Dr. Kestler fused the knee in July 1956 because of continued pain in the knee. He has told me that the knee is stable but that fusion is fibrous.

COMMENT

It is apparent that an operation on a neuropathic joint is quite unlike an operation on a joint that is not neuropathic. There are more risks for the patient. The first is death. In January 1958 a man aged 56 died suddenly three days after a well-done Charnley operation for fusion of a Charcot knee joint, seemingly of an embolus, when his postoperative course had seemed easy and secure. (The singular aspect of this early arthropathy was the slicing away of the posterior part of the medial condyle.) More to be feared is the infection to which the Charcot knee is prone after either aspiration or operation.

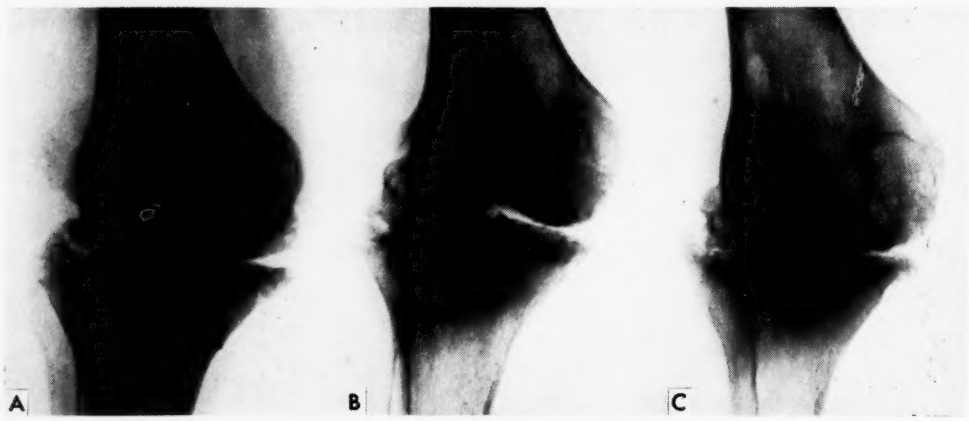


Fig. 8.—*A*, preoperative anteroposterior roentgenogram taken on Dec. 27, 1955, of left Charcot knee. *B*, postoperative anteroposterior roentgenogram taken Feb. 28, 1956, showing patella implanted into lateral tibial condyle to correct valgus. *C*, anteroposterior roentgenogram taken May 8, 1956. Patella is not countersunk but is not fully joined to its bed. *(I am indebted to Dr. O. C. Kestler, who permitted me to reproduce these roentgenograms.)*

Careful asepsis and the use of antibiotic medication should lessen the possibility of infection which, if uncontrolled, demands amputation. And always, the pathophysiologic block to success is lack of feeling within the joint. The response of the joint to operation is made more certain if the adverse effect of weight is realized.

Operation should be done early, for fusion or reconstruction in the presence of advanced neuropathic disease of joints achieves little or nothing.

SUMMARY

The Charcot joint is made insensitive by the loss of proprioception. The destruction of the medial tibial condyle follows as the effect of the shear component of the weight force, whose maximal angle is 45 degrees. The differential elements of the weight force wreak wide havoc, predominantly on the concave medial tibial condyle. There is limited change on the convex medial femoral condyle. Reconstruction of the Charcot joint should break up the vicious circle of destruction that results in the vicious circle of deformity, further destruction and further deformity. Yet the reconstructed joint itself lacks proprioception, so that it too may become neuropathic unless it is protected against shear.

A vitallium mold replaced the fragmented medial tibial condyle of the Charcot knee joint in varus, with the impression that the indestructible material would resist the weight force after correction of the deformity. The operation was done twice, once in 1944 and again in 1945, before antibiotic materials became easily available. Each operation failed. In the first patient the wound became infected, and the mold was removed two months after its implantation. The persistence of infection made midthigh amputation necessary five months later. In the second patient the advance of neuropathy in the left knee had been stayed by synovectomy almost two years before. The distal part of the wound in the right knee, into which the mold was implanted, failed to heal, and the probe, passed through the sinus, led to the metal. Secondary infection of the synovial fistula made removal of the mold necessary sixteen months after it had been put in.

More recently, late in 1955 and early in 1956, the patella was used to lift up the depressed lateral tibial condyle in 2 patients with severe valgus deformity of the Charcot knee. In the first of these cases a second operation had to be done some months after the patellar implantation, to tighten the internal lateral ligament. One operation failed (pain), and fusion had to be done five months later. In the other case more than three years have passed and the operation is reasonably successful, for it has done what it set out to do. The joint is supported by a brace. It is stable in full extension. The disease has not advanced. Active extension is allowed to 170 degrees and active flexion to 90 degrees.

Reconstruction does not provide the full security of fusion, but it stays the disease if infection does not follow the operation or come after aspiration. It should be done earlier rather than later when retention of motion is desired, or in one knee if both are afflicted with neuropathic disease.

The use of the patient's own patella is probably better than the use of a foreign substance, such as a metal or plastic mold, which the neuropathic joint does not seem to tolerate.

ZUSAMMENFASSUNG

Das Charcotsche Gelenk ist durch den Verlust des Muskelsinns unempfindlich geworden. Die Zerstörung des medialen Kondylus der Tibia erfolgt unter dem Einfluss des scherenden Anteils der Gewichtsbelastung, dessen grösster Winkel 45 Grad

beträgt. Die differentialen Elemente der Gewichtsbelastung führen zu umfangreichen Verheerungen besonders an dem konkaven medialen Kondylus der Tibia. Die Veränderungen am konvexen medialen Kondylus des Oberschenkelknochens sind beschränkt. Die Wiederherstellung des Charcotschen Gelenks sollte den circulus vitiosus von Zerstörung, die zur Deformierung und von Deformierung, die zur Zerstörung führt, unterbrechen. Das wiederhergestellte Gelenk jedoch hat auch keinen Muskelsinn, sodass es selbst auch neuropathisch werden kann, wenn es nicht vor dem Scherungseffekt geschützt wird.

In einem Charcotschen Kniegelenk in Varusstellung wurde der gebrochene mediale Kondylus der Tibia durch einen Vitalliumabguss ersetzt. Man dachte sich, dass das unzerstörbare Material nach Verbesserung der Deformierung den Gewichtskräften widerstehen würde. Die Operation wurde an zwei Patienten ausgeführt, einmal im Jahre 1944 und das zweite Mal im Jahre 1945, d.h. zu einer Zeit wo Antibiotika leicht erhältlich waren. In beiden Fällen versagte die Operation. Bei dem ersten Mann kam es zu einer Infektion der Wunde, und der Abguss wurde zwei Monate nach seiner Einpflanzung wieder entfernt. Das Weiterbestehen der Infektion machte fünf Monate später eine hohe Amputation erforderlich. Bei dem zweiten Mann war das Fortschreiten der neuropathischen Erkrankung des linken Kniegelenks durcs Resektion der Synovia fast zwei Jahre vorher zum Stillstand gebracht worden. Am rechten Knie, in das der Abguss eingepflanzt war, blieb die Heilung des distalen Abschnittes der Wunde aus, und eine Sonde, die durch die Fistel eingeführt wurde, führte zum Metall. Sekundäre Infektion der Synovialfistel machte die Entfernung des Abgusses 16 Monate nach seiner Einführung notwendig.

In jüngerer Zeit, nämlich Ende 1955 und Anfang 1956, wurde zur Hebung des herabgerückten lateralen Kondylus der Tibia bei zwei Kranken mit schwerer Valgusdeformierung eines Charcotschen Kniegelenks die Kniescheibe benützt. Bei dem ersten dieser beiden Fälle musste einige Monate nach Einpflanzung der Kniescheibe eine zweite Operation zur Spannung des inneren lateralen Ligaments vorgenommen werden. Die Operation war erfolglos (Schmerzen), und fünf Monate später musste eine Fusion des Gelenks ausgeführt werden. In dem anderen Fall sind drei Jahre seit dem Eingriff vergangen, und die Operation ist relativ erfolgreich, weil sie zu dem beabsichtigten Resultat geführt hat. Das Gelenk wird durch eine Schiene unterstützt. Die Erkrankung ist nicht fortgeschritten. Das Gelenk ist stabil bei voller Streckung. Aktive Streckung kann bis zu 170 Grad und aktive Beugung bis zu 90 Grad erfolgen.

Die Wiederherstellung des Gelenks gibt nicht die volle Sicherheit einer Fusion, aber sie bringt die Erkrankung zum Stillstand, sofern keine Infektion nach der Operation erfolgt. Der Eingriff sollte lieber frühzeitig als zu spät erfolgen, wenn Erhaltung der Bewegungsfähigkeit erwünscht ist, oder sollte an einem Knie vorgenommen werden, wenn beide Seiten neuropathisch erkrankt sind.

Die eigene Kniescheibe des Patienten eignet sich wahrscheinlich besser zur Einpflanzung als ein künstliches Material wie metallische oder Kunststoffabgüsse, die das neuropathische Gelenk nicht gut zu vertragen scheint.

SUMARIO

A articulação de Charchot torna-se insensivel pela perda da propriocepção. A destruição do condilo tibial medial segue-se como efeito do componente do peso cujo ângulo máximo é de 45 graus. Os elementos diferenciais executam grave dano predominantemente na concavidade do condilo ti-

bial medial. A reconstrução dessa articulação deve quebrar o circulo vicioso da deformidade, evitando maior destruição e portanto maior deformidade. Mesma quando reconstruida a articulação permanece sem sensibilidade proprioceptiva de maneira que se torna neuropatica a menos que seja protgida contra o desvio. Um molde de vitalio substitue o condilo medial fragmentado da articulação do joelho em varus. Esse material pode resistir a força de destruição apos a correção da deformidade. A operação foi praticada duas veses em 1944 e 1945 antes da era de facilidades do uso de antibioticos. Ambas falharam. No primeiro caso houve infecção da ferida e a protese removida two meses depois. Essa infecção obrigou a uma amputação alta cinco meses mais tarde. No segundo paciente a neuropatia foi detida no joelho esquerdo por uma sinovectomia dois anos antes. Não houve cicatrização da porção distal da ferida que fistulisou. A fistula sinovial obrigou a remoção da protese 16 meses mais tarde. Mais recentemente, em 1955 e 1956 a rótula foi utilisada para preencher o espaço deixado pelo côndilo tibial lateral em dois pacientes com valgus grave do joelho. No 1° desses dois casos a segunda operação foi feita alguns meses depois do enxêrto de rótula para tensionar o ligamento lateral interno. Uma operação falhou (dor) e a artrodese foi feita cinco meses mais tarde. No outro caso depois de três anos o resultado da operação ainda era rasoavel. A molestia não progrediu. É estavel na extensão completa. A extensão ativa vai a 170° e a flexão ativa á 90°.

A reconstrução não dá a mesma segurança que a fusão mas detem a molestia se não houver infecção. Deve ser praticada precocemente, quando há ainda desejo de obter movimento ou em um dos dois joelhos se ambos são neuropaticos. O uso de rotula antogena é provavelmente melhor que uma protese, metalica ou plástica, porque a articulação neuropatica parece não tolerar.

RÉSUMÉ

L'arthropathie tabétique est rendue indolore par la perte della proprioception. La destruction de la tubérosité interne du tibia résulte de l'effet des forces composées du poids, dont l'angle maximum atteint 45°. Les éléments différentiels des forces donnent lieu à des destructions étendues, surtout de la partie concave de la tubérosité interne du tibia. Il y a peu de modifications de la partie convexe du condyle interne du fémur. Le traitement de l'arthropathie tabétique devrait rompre le cercle vicieux de destruction aboutissant à un cercle vicieux de déformation (plus la destruction augmente, plus la déformation s'accentue). Cependant l'articulation reconstruite manque elle-même de proprioception, de sorte qu'elle peut, ella aussi, devenir neuropathologique, à moins qu'elle soit protégée.

L'articulation du genou fragmenté en varus a été remplacée par un moulage en vitellium. Il semblait que cette substance indestructible résisterait à la force du poids une fois la déformation corrigée. L'opération a été pratiquée deux fois, en 1944 puis en 1945. Cet fut un échec dans les deux cas. Dans le premier, la plaie s'est infectée, et le moulage a dú être retiré deux mois après. La persistance de l'infection a nécessité une amputation à mi-hauteur cinq mois plus tard. Dans le second cas le développement de la neuropathie du genou gauche avait été stabilisé par une synovectomie près de deux ans auparavant. La partie distale de la plaie du genou droit à l'endroit où avait été introduit le moulage, ne s'est pas cicatrisée et il a fallu le supprimer après seize mois en raison d'une infection secondaire de la fistule synoviale.

Plus récemment, à la fin de 1955 et au début de 1956, l'auteur a utilisé la rotule pour corriger la dépression de la tubéro-

sité externe du tibia chez deux malade atteints de déformation en valgus grave (arthropathie tabétique). Le premier cas a nécessité une deuxiéme opération quelques mois après l'implantation rotulienne, afin de resserrer le ligament latéral interne. Une opération a échoué (douleurs) et il a fallu procéder à une arthrodèse cinq mois plus tard. Dans l'autre cas, qui date de plus de trois ans, l'opération a donné les résultats raisonnablement satisfaisants que l'on pouvait en attendre. L'articulation est soutenue par une sorte d'amarre, elle est stable en extension totale. L'affection n'a pas progressé. L'extension active est possible à 170°, la flexion active à 90°.

La reconstruction ne donne pas la sécurité totale de l'arthrodèse, mais elle stabilise l'affection si l'operation n'est pas suivie d'une infection. L'intervention devrait être plutôt précoce lorsqu'on vise à une conservation de la mobilité, et elle devrait être pratiquée sur un seul genou lorsque les deux sont atteints d'une affection neuropathogène.

Il est probablement préférable d'utiliser la rotule du patient plutôt qu'une substance synthétique telle que le moulage plastique ou métallique, que l'articulation atteinte ne paraît pas tolérer.

RESUMEN

La articulación de Charcot se hace insensible por pérdida de la propioconcepción. La destrucción del cóndilo medio tibial es consecuencia de un componente de el peso y la posición de 45°. Los elementos diferenciales del peso y la fuerza determinan un gran trastorno especialmente en el cóndilo tibial interno. La reconstrucción de la articulación de Charcot dene romper el círculo vicioso que resulta de destrucción, deformidad y mayor destrucción.

No obstante la articulación reconstruída está falta también de proporción y se volverá también neuropática salvo que se la proteja adecuadamente. Para suplir el cóndilo interno destruído se coloca un molde de vitallo con la rodilla en varus. La operación ha sido efectuada dos veces una en 1.944 y otra en 1.941 antes de disponer fácilmente de los antibióticos. Ambas operaciones fracasaron. En el primer caso la herida se infectó y el molde hubo de ser eliminado dos meses después de su colocación y la persistencia de la infección obligó a la amputación del muslo 5 meses después. En el segundo caso el avance de la neuropatía en la rodilla izquierda fué detenido por rinoviectomía casi dos años antes. La parte distal de la herida de la rodilla derecha y a traves de la cual se había colocado la prótesis no cicatrizó y con una sonda introducida en la fístula se tocaba la pieza metálica. Una infección secundaria obligó a eliminar la pieza de vitalio 16 meses después.

Más recientemente, en 1.955 y 56, se ha empleado la rótula para alzar el cóndilo deprimido en la articulación de Charcot con valgus acentuado. En el primero de estos casos fué precisa una nueva operación algunos meses después de la implantación rotuliana para poner tenso el ligamento lateral interno. En otra operación el fracaso por dolor, obligó a una fusión cinco meses más tarde. En el tercer caso han pasado más de 3 años y la operación satisfactoria ha dado todo cuanto se esperaba de ella. La articulación se ayuda con una rodillera, la enfermedad no ha avanzado, hay estabilidad y extensión completa, y la movilidad activa oscila entre 90° y 170°.

La reconstrucción no es tan absolutamente segura como la fusión pero detiena la enfermedad si la infección no complica la operación. Debe ser hecha lo antes posible si lo que se desea es la inmovlización o en una rodilla cuando ambas están afectadas de neuropatía. El uso de la rótula es probablemente menor que una sustancia extraña, metálica o plástica que parece

ser no es tolerada por la articulación enferma.

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Surgical Aid for Arthritis of the Hand and Elbow*

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ALTHOUGH the scope of surgical treatment of the arthritic is limited, its role is becoming more clearly defined and presents a stimulating challenge with regard to deformities of the hand. During the acute stage of the disease a course of splinting consisting of a balance between motion and rest, together with some form of local heat, is pursued. In the chronic stage of the disease it must be emphasized that a patient with a hand that functions well and has no severe pain is generally not a candidate for corrective surgical intervention, despite the rheumatoid deformities in the peripheral joints.

Elbow.—Surgical treatment of the elbow region can be divided into nerve transposition and corrective bone operations.

Paresis of the ulnar nerve can follow rheumatoid degeneration of the elbow. Just as in median nerve compression at the wrist there also exists ulnar nerve compression at the elbow, which is chiefly due to the swelling within the joint capsule and the periarticular tissues, and occasionally to the incongruous surfaces within the medial condylar groove. Simple transposition of the ulnar nerve yield rewarding results. Obviously, the ulnar nerve should be transposed early, before irreversible muscle changes in the hand have occurred.

As was suggested by Smith-Petersen, there is often an *anterior displacement of the head of the radius,* with destruction of the articular surfaces, which distorts its normal relation to the capitellum. The dislocation may be due to protective muscle spasm of the biceps. Smith-Petersen advised resection of the radial head when the arthritic involvement is localized to this area.[1] The principle has been followed and resection of the radial head as a procedure preliminary to arthroplasty of the elbow has been utilized. An improvement in supination-pronation before the arthroplasty is obtained by this procedure.

The technic employed for arthroplasty of the elbow has included the use of a full thickness skin graft after the humeral condyles have been reshaped and the olecranon fossa hollowed out. The skin graft must be placed under taut tension over the distal end of the humerus (Fig. 1) to prevent inclusion cysts, which may otherwise develop and jeopardize the end result.

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It is extremely rare for a quiescent rheumatoid disease to be aggravated by an orthopedic surgical procedure, and this medical myth should be exploded. Despite the nature of the disease, the manner in which rheumatoid hands have tolerated surgical therapy has been surprising, and the results procured represent greater function and decreased pain. A table indicating the nature of the procedure and its indications is presented.

The Wrist.—A common arthritic deformity observed at the wrist is *subluxation of the distal portion of the ulna.* Often this is relatively painless. When pain occurs, however, especially in full supination, resection of the distal portion of the ulna almost always relieves the symptoms. Cregan advised resection of the distal 2½ inches (6.2 cm.) of the ulna, stating that he gains function in pronation-supination without losing significant strength. However, a loss of power of the pronator quadratus will result, however, and subluxation of the distal end of the ulna may cause a significant cosmetic disadvantage after such an extensive resection. In the present series the rotatory restriction after resection of the ulnar head was due either to radiocarpal arthritis or to changes involving the radial head.

Arthrodesis of the wrist is performed for pain from radiocarpal arthritis and also for irreversible malposition. The technic employed is to remove the ulnar head and use it as a graft after removing the remnants of cartilage from the radiocarpal joint, the proximal carpal row and the area around the capitate. It has not been necessary to extend the fusion to the carpometacarpal joints, as these rarely cause pain from arthritic involvement.

Median Nerve Decompression.—In support of Onne, it has been observed that the most common cause of nonspecific synovial changes about the wrist is early arthritic involvement, and frequently this process is accompanied by compression of the median nerve.[2] This may result in paresthesia of the median nerve swelling within the flexor tendon sheaths at the wrist and weakness of the thenar muscle. Cregan[3] reported that division of the anterior carpal ligament and excision of the flexor sheath above the wrist gave uniformly excellent results. This procedure is useful, but it is the decompression of the median nerve which is responsible for the relief

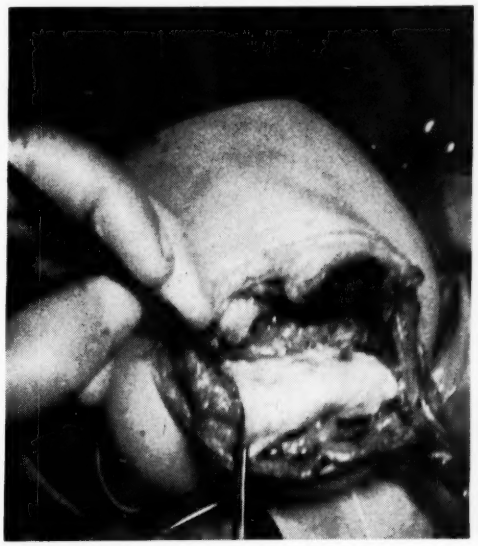

Fig. 1.—Cutaneous arthroplasty of elbow, showing reshaped olecranon and skin graft being tightly drawn over humeral condyles.

of pain. Total excision of the synovial tissue in this area, however, diminishes the likelihood of recurrence of the swelling. The nebulous and hazy diagnosis of "idiopathic acroparaesthesia" for this condition, according to some works on physiotherapy, has resulted in irreparable and irreversible damage to the median nerve. The subsequent lack of adequate treatment should be severely condemned. Early decompression of the median nerve is simple, direct and effective treatment.

Soft Tissue Involvement Within The Digits.—It is likely that *tenovaginitis* as exemplified by De Quervain's disease and the adult type of trigger finger is often related to an early or localized process involving only synovial tissue at first. In many cases in which these are present, well-defined arthritic symptoms develop at a later date. The stenosing synovial tissue has been excised whenever the symptoms warranted it. In the early stages of trigger finger, prior to the development of fibrotic

stenosis, injections of hydrocortisone into the digital sheath have been effective.

Some authors have stated that painful *nodules on the flexor tendons* should not be removed, since over a period of one year the symptoms will spontaneously recede.[3] This could not be corroborated, and it has been observed that these nodules cause considerable pain, interfere with a strong grip and occasionally result in a regettable secondary deformity. They should be excised early, therefore, with regional block anesthesia. These rheumatoid nodules may infiltrate into tendon tissue and cause rupture of the tendon (Fig. 2).

REPORT OF CASE

A 43-year-old-woman had a tender and painful 3 cm. nodule overlying the volar aspect of the metacarpal-phalangeal area of the index finger for three years. The nodule increased in size and became more painful during this interval, so that the patient's grip was poor and she often dropped objects. There was a previous history of a snapping finger in this area. The patient exhibited pain on palpation of the third and fifth phalangeal and interphalangeal joints of the same hand.

At operation, extensive involvement of the volar sheath of the index finger was noted, with a large, hard nodule lying on the sublimis tendon and invading the tendon at the M-P level as well as at the decussation. The sublimis tendon had been destroyed by the nodule and was on the point of rupture. The sublimis displayed moderate swelling, with an area of constriction at the point of obstruction which was the site of the previous snapping finger. The profundus was relatively free of pannus involvement. To prevent invasion of the profundus tendon, therefore, and to permit gliding motion of the tendon, the sublimis was excised. The report of the pathologist confirmed invasion of the tendon and tendon sheath by the rheumatic process.

Drummer's Palsy.—This results from spontaneous rupture of the extensor pollicis longus tendon at Lister's tubercle. Primary suture of the tendon rupture often results in a secondary division at the site of anastomosis, owing to the ischemia within the tendon. The treatment is to use the extensor carpi radialis longus (not the extensor carpi radialis brevis, which is a strong wrist stabilizer) or the extensor indicis proprius as the motor. The wrist extensor is favored by this clinic, since use of the extensor indicis proprius occasionally results in loss of full extension of the index finger and some impairment of its independent action.

Rupture of the Extensor Digitorum Communis.—This is a rare injury. When caused by rheumatoid arthritis, it is often produced by friction of the tendon against the rough surface of the deformed ulnar head. Resection of the ulnar head and either tendon transplant or a graft have been effective.

Rupture of the Flexor Pollicis Longus.—When this occurs at the level of the

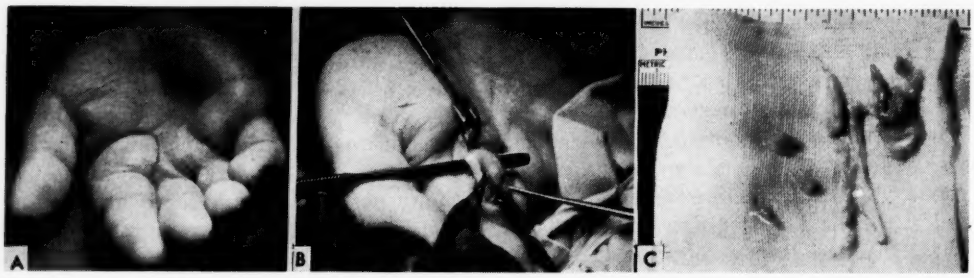

Fig. 2.—*A*, painful rheumatoid nodule of three years duration outlined in ink. *B*, nodule on flexor sublimis. *C*, appearance after excision of sublimis. Upper left is nodule, with panus tissue at right. Note marked involvement of sublimis at decussation.

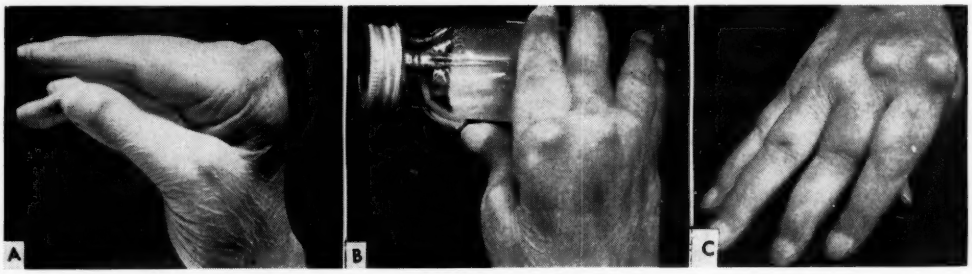

Fig. 3.—*A*, intrinsic plus deformity, or "opera glass" hand. *B*, inadequate grip due ulnar and volar subluxation. *C*, ulnar and volar subluxation.

flexor sheath it should be treated, not by resection of the diseased tendon and primary suture, but by resection with a free graft transplant. Moberg[4] has not found it necessary to place the proximal suture of the graft above the wrist as some authors advise; he places it in the thenar area. This simplifies the technic of the procedure, and in his hands the results have been rewarding.

Intrinsic Muscle Spasm.—According to Fowler, this is often the first manifestation of rheumatoid arthritis of the hands. Bunnell[5] has demonstrated this condition by the inability of the patient to flex the interphalangeal joints with the metacarpal-phalangeal joints extended. It is treated by release of the intrinsic muscle tendon unit through section of the intrinsic tendons at the metacarpal-phalangeal joint or the lateral bands opposite the proximal interphalangeal joints (Goldner[6]). The range of surgical treatment here is limited, however, and operation is reserved for highly selected cases. In a case of early intrinsic muscle spasm operation is not necessary, and in a case of sever involvement it is often not effective because of joint changes and fixed contractures. It is far better to prevent intrinsic contractures by active exercises and splinting before the spasm evolves into a severe deformity.

Dislocation of Digits.—Thumb: Dislocations occur at the metacarpal or the interphalangeal joint. This often results in adduction deformity of the thumb, with resultant inability to oppose. When this disability is irreversible or instability is present, an arthrodesis of either the interphalangeal or M-P joint is done. The results of more than 60 such fusions performed by Vainio[7] have been encouraging, and the power of the thumb is greatly increased. Caution must be exercised, however, before performing fusion of either distal joint, to make sure some power of abduction exists at the carpometacarpal joint because arthrodesis may make it impossible for the patient to open his grip and thereby prevent him from holding a glass or gripping a crutch.

Metacarpal-Phalangeal Dislocations of the Digits.—This is a common deformity accompanying the intrinsic plus or "opera glass" deformity. The initial process is swelling and distention of the metacarpal-phalangeal joint with resultant overstretching of the capsule and ligaments. This often results in volar subluxation of the metacarpal-phalangeal joint, with a corresponding ulnar drift. There is usually ulnar subluxation of the extensor tendon into the knuckle grooves. The indication for surgical therapy is a grip so deformed and painful that function is not possible (Fig. 3).

An arthroplasty of the metacarpal-phalangeal joint devised by Vainio is performed by excising the metacarpal head and pannus tissue. The radial collateral lig-

ament is sutured to the metacarpal neck to help correct the ulnar drift. The extensor tendon is divided, and the distal stump is utilized for the volar subluxation. The shortened extensor tendon is then placed in the dorsal hood by overlapping the extensor expansion in such a way as to reinforce the correction of the ulnar displacement. In Vainio's series of 34 operative cases he observed that about 50 per cent of the patients have some tendency toward a degree of ulnar subluxation, but correction of the volar subluxation remains improved. Generally, the pain is considerably reduced. In view of the advanced deformity of such hands, a considerable increase in function and decrease in pain are generally obtained, and a fairly strong grip is acquired. The number of patients who have requested this procedure on the opposite hand when the process is bilateral has been encouraging.

Dislocation of the Interphangeal Joints.—This is represented in its advanced stage by the "buttenhole deformity" of the proximal interphalangeal joint. It is caused by distention of the capsule resulting in volar displacement of the lateral bands. When advanced, the proximal interphalangeal joint is fixed in flexion. Arthroplasty on this joint has limited value and should be restricted to selected cases. When motion is markedly decreased, arthrodesis of the proximal interphalangeal joint in 30 degrees of flexion is performed, and this frequently relieves an otherwise painful grip. Moberg,[4] in a review of 150 finger arthrodeses stated that the quickest and most reliable method of fusion is obtained by denuding the articular surface of carti-

Surgical Treatment of Arthritis of the Hand and Elbow

	Operative Procedure	*Indications*
Elbow	1. Translocation of ulnar nerve	Pain Ulnar nerve paresthesia with palsy
	2. Resection of radial head	Pain Limited rotation
	3. Arthroplasty of elbow joint	Pain Advanced bone involvement
Wrist	1. Decompression of median nerve	Pain Median nerve paresthesia Thenar weakness
	2. Excision of synovial sheath on flexor or extensor surfaces	Swelling with pain, De Quervain's disease
	3. Wrist arthrodesis	Painful, unstable wrist with mobile fingers
	4. Resection of ulnar head	Pain on rotation Subluxation of ulnar head
Fingers	1. Arthrodesis of thumb	Painful dislocation or instability
	2. Excision of stenosing tissue with flexor sheath	Stenosis of tendon resistent to conservative treatment
	3. Excision of flexor tendon nodules	Painful grasp
	4. Arthroplasty of proximal interphalangeal joints	Dislocation with painful motion (only in selected cases)
	5. Arthrodesis of interphalangeal joints	Fixed, painful dislocation
Tendons	1. Tendon transfers or tendon graft	Drummer's palsy Rupture of flexors Rupture of extensors
	2. Section of intrinsic tendons or lateral bands	Only in selected cases of intrinsic contracture

lage, shaping it to obtain 30 degrees of flexon and then inserting a quadrilateral bone graft into the intramedullary portion of the proximal and middle phalanges.

Arthrodesis of the distal interphalangeal joint is preferred to the unrewarding results of arthroplasty at this level.

Author's Note: Grateful acknowledgment is made to Dr. K. Vainio of the Rheumatic Hospital in Heinola, Finland, and to Prof. E. Moberg of Sahlgrenska Sjukhuset in Goteborg, Sweden. It is their experience and knowledge of hand surgery upon which most of these procedures are based.

CONCLUSIONS

Il est extrêmement rare qu'une affection rhumatoïde chronique soit aggravée par une opération orthopédique, et il est temps de détruire ce mythe. Malgré la nature de l'affection, la façon dont les mains rhumatisantes ont toléré un traitement chirurgical s'est révélée surprenante et les résultats obtenus montrent une augmentation de la fonction ainsi qu'une diminution des douleurs. L'auteur présente un tableau indiquant la nature de l'opération et ses indications.

SCHLUSSFOLGERUNGEN

Es ist ausserordentlich selten, dass eine ruhende rheumatische Erkrankung durch einen orthopädischen chirurgischen Eingriff verschlimmert wird, und diese medizinische Fabel sollte ein für alle Mal aus der Welt geschafft werden. Trotz der Eigentümlichkeiten der Erkrankung haben rheumatische Hände chirurgische Behandlung erstaunlich gut vertragen mit dem Resultat besserer Funktion und verminderter Schmerzen. Die Art und die Indikationen des Verfahrens werden in Form einer Tabelle dargestellt.

RESUMEN

Es extremadamente raro que una enfermedad reumática latente sea agravada por una intervención quirúrgica, y este mito debe ser destruído. A pesar de la naturaleza de la enfermedad, el modo como algunas manos reumatoides han tolerado la cirugía ha sido sorprendente y los resultados obtenidos de una mejor función y una mejoría del dolor. Se presenta un cuadro indicando la naturaleza del procedimiento y sus indicaciones.

CONCLUSOES

É extremamente raro uma afecção reumatismal silenciosa possa se agudisar pela ação de metodos cirurgico e ortopedico. Á despeito da natureza da molestia é surpreendente como as mãos reumáticas toleram os metodos cirurgicose concedem resultados muito bons sobre a função e a dor.O autor apresenta um quadro indicando os processos cirurgicos e suas indicações.

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Diffuse Infiltrating Adenocarcinoma of the Rectum

Report of Three Cases

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IN 1854 Brinton applied the term "linitis plastica" to a condition in which the stomach showed a peculiar thickening of the entire wall. He considered it an inflammation of the areolar tissue ensheathing the vessels. Lietaud in 1779 may have recognized the condition, although Andral is credited with publishing the first case report in 1829.

The diffuse infiltrating type of carcinoma was long considered limited to the stomach, constituting the classical "leather bottle" variety. Indeed, in the great majority of cases it has a predilection for this organ, but the same histologic features have been demonstrated in the small bowel, the breast, the gallbladder, the colon and the rectum.

An organ attacked by diffuse infiltrating adenocarcinoma shows thickening of the wall, which produces varying degrees of contraction. The mucosa usually appears intact, yet may present a pebbled appearance without ulceration. On occasion a few excoriations may be noted. The extent of involvement ranges from a narrow, annular contraction to an advanced stage making the wall inflexible. Microscopically the fibrous tissue is arranged more or less in a network, a pattern usually most pronounced in the submucosa. Scattered among the fibrotic areas are carcinoma cells with large nuclei, which often have

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Diffuse infiltrating adenocarcinoma presents great inflammatory changes that present the aspect of a benign process. It is extremely rare as a primary growth in the rectum, but it is important to recognize the fact that atypical infiltrating lesions of the rectum may be metastatic.

Three cases are presented in which the primary location of the tumor in the rectum was established.

Because of the similarity of this condition to lymphogranuloma, venereum, efforts should be made toward correct diagnosis, since treatment and prognosis of the two conditions are different.

a vesicular appearance. Many of the infiltrating carcinomatous cells do not reach complete development, failing to form glandular structures; nevertheless, they maintain the ability to produce a secretion that causes compression of the nuclei, giving rise to the typical signet-ring type of cell.

In extremely rare instances invasion of the rectum by diffuse infiltrating adenocarcinoma may be due to a primary growth or may occur as a secondary lesion arising in some other organ, frequently the stomach. Signet-ring cells in carcinoma of the breast, gallbladder, stomach or colon have a tendency to metastasize to the ovaries, the rectovesical pouch and the rectovaginal septum, but even more commonly to the peritoneum.

Presumably, metastasis follows the lymphatic or hematogenous channels and is not the usual form of transceolomic spread observed in rectal shelf or rectovesical pouch implants.

Coe, in 1931, reported a case in which there was involvement of both flexures of the colon as well as the rectum. In 1936 Dixon, Stevens and Arnold[1] cited 6 cases, in 1 of which there was a typical lesion of the rectum. These authors collected 37 cases in which there was involvement of one or more portions of the gastrointestinal tract. Saphir and Parker[2] published a report of 3 patients with the linitis plastica type of carcinoma, in 2 of whom the process was located in the rectum. In a later publication[3] they recorded 4 cases of primary diffuse infiltrating adenocarcinoma of the colon, with confirmation of their observations at necropsy. Three additional cases of metastasis to the rectum were cited by Schied and Crile.[4]

In a review of 1,778 patients with carcinoma of the rectum and colon in the service of Dr. Bacon, only 3 patients showed the characteristics of a primary tumor of this kind. Their cases are here reported.

REPORT OF CASES

CASE 1.—I. C. T., a 63-year-old Negro man whose principal complaint was of bearing-down gripping pain accompanied by a desire to defecate, was admitted to the hospital on Nov. 24, 1948. The symptoms were of two years' duration and associated with constipation. There had been loss of almost 10 pounds (4.5 Kg.). There were no gastric symptoms. Examination revealed stricture of the anal canal and rectum that allowed entrance of the small finger only. The rectal mucosa was finely granular and thick. A transverse colostomy solved the problem of intestinal obstruction that developed four days later. Roentgen studies demonstrated pronounced narrowing of the rectum.

Lymphopathia venerea was suspected clinically, but the antigenic Frei test gave negative results. Biopsy disclosed a chronic inflammatory reaction.

Rectal resection with preservation of the sphincter mechanism was carried out. The specimen showed the distal 8 cm. of the rectum to be extremely narrowed; the rectal walls were thick with fibrosis extending to the outer layers and the perirectal fatty tissue.

Microscopic examination revealed diffuse infiltrating adenocarcinoma with metastasis to the lymph nodes. The mucosa was not involved by the process, but neoplastic elements were seen in the remaining layers.

The patient reentered the hospital five months later, where he succumbed to intestinal obstruction caused by malignant invasion of the small bowel. There was no evidence of malignant change in the stomach.

CASE 2.—M. S., a 63-year-old white man, was admitted to the hospital in November 1958 because of pain on defecation of two weeks' duration, accompanied by a sensation of rectal pressure. Four cm. above the anorectal line, examination disclosed a roughly rugose elevation of the mucosa, which was somewhat reddened; the elevation extended upward 2 cm. and surrounded the mucosa of the bowel in annular fashion. The first biopsy showed normal rectal mucosa, but a specimen taken from a deeper level established the diagnosis of malignant neoplasm. After abdominoperineal excision the pathologic diagnosis was diffuse infiltrating adenocarcinoma of the rectum with infiltration of the perirectal tissue. Six months after the operation, microscopic studies of the peritoneal fluid disclosed malignant cells.

CASE 3.—A. S., a 51-year-old white woman, complained of change in bowel habit and constipation of one year's duration and reported having experienced the sensation of incomplete bowel movements two months before admission. Digital examination detected a firm, fixed ring encircling the entire rectum, at a level about 7 cm. from the anal verge. The mucosa appeared firm. Normal rugations were absent. Biopsy tissue showed carcinoma infiltrating the submucosa. Abdominoperineal excision was carried out. The pathologic diagnosis was diffuse infiltrating adenocarcinoma of the rectum with extension to the lymphatics and serosa.

COMMENT

In establishing the diagnosis of diffuse infiltrating adenocarcinoma of the rectum, special attention should be paid to the presence or absence of gastric symptoms, and the stomach must be ruled out as a primary site, since this type of carcinoma constitutes a diagnostic problem in itself.

The symptoms reported focus on changes in bowel habit. Patients seek attention because of constipation, incomplete defecation and rectal discomfort. In these 3 cases bleeding was not apparent, and only 1 patient reported loss of weight.

Biopsy specimens taken by the usual methods may show normal rectal mucosa or inflammatory changes, but specimens taken from deeper levels, including the submucosa and surrounding tissues, show the typical carcinoma cells. The diagnosis of malignant tumor may be possible only after examination of the entire specimen.

Differentially, lymphogranuloma venereum must be kept in mind, for it may have invaded the rectal wall. A lesion with indurated walls, apparently normal rectal mucosa and negative reaction to the Frei test suggests a malignant process.

One must be on the alert to recognize a secondary growth, metastasizing from the stomach, that may simulate a primary lesion of the rectum. Too often the primary source remains asymptomatic.

Treatment should follow the same principles advocated for rectal carcinoma, that is, excision and lymphadenectomy when the patient offers a good surgical risk.

SCHLUSSFOLGERUNGEN

Diffus infiltrierende Adenokarzinoma des Mastdarms können erhebliche entzündliche Veränderungen aufweisen, die der Geschwulst das Aussehen einer gutartigen Veränderung geben. Als primäre Geschwulst kommt dieser Mastdarmtumor äusserst selten vor; es ist aber wichtig zu wissen, dass atypische infiltrierende Erkrankungen des Mastdarms metastatische Geschwülste sein können.

Es wird über drei Fälle berichtet, bei denen der Primärsitz der Geschwulst im Mastdarm nachgewiesen wurde.

Da diese Erkrankung Ähnlichkeiten mit dem venerischen Granulom aufweist, sollte nichts unversucht bleiben, zu einer richtigen Diagnose zu gelangen; denn die Behandlung und Prognose der beiden Erkrankungen sind verschieden.

CONCLUSIONS

L'adénocarcinome diffus infiltrant entraîne d'importantes modifications inflammatoires qui présentent l'aspect d'un processus bénin. Il est extrêmement rare en tant que tumeur primaire dans le rectum, mais il est important de savoir que des lésions infiltrantes atypiques du rectum peuvent être métastatiques.

Trois cas sont présentés où il a été établi que la tumeur primaire était à point de départ du rectum.

En raison de la ressemblance de ces cas avec la maladie de Nicolas-Favre, les efforts doivent être concentrés en vue d'un diagnostic exact, le traitement et le pronostic de ces deux états étant différents.

CONCLUSIONE

Il carcinoma diffuso infiltrante presenta spesso fenomeni infiammatori che gli danno l'aspetto di un processo benigno.

Questo tipo di tumore é estremamente raro nel retto, Ne vengono presentati tre casi; e' necessario compiere uno sforzo per giungere ad una diagnosi corretta, dal momento che le lesioni sono molto simili a quelle del linfogranuloma venereo, mentre la prognosi e la terapia delle due lesioni sono completamente differenti.

CONCLUSOES

O carcinoma difuso infiltrativo apresenta grandes modificações inflamatorias que dão o aspecto de lesão benigna. No reto é extremamente raro como processo primitivo mas é importante lembrar que lesões infiltrativas atipicas podem ser metastaticas. Apresenta tres casos em que a localisação primitiva foi no reto. No diagnostico diferencial lembra a similaridade ao linfogranuloma pelo que o diagnostico deve ser bem estabelecido considerando-se que o tratamento e prognostico são diversos.

CONCLUSIONES

El adenocarcinoma infiltrante difuso presenta grandes alteraciones inflamatorias que le dan el aspecto de proceso benigno. Se trata de una afección poco frecuente como proceso primitivo en el recto, pero es importante saber que lesiones infiltrantes atípicas rectales pueden ser metastáticas.

Se presentan tres casos en los que se puede demostrar la implantación primaria en el recto. Deben implearse todos los medios diagnósticos para un diagnóstico correcto por su gran similitud con el linfo granuloma venereo ya que el pronóstico y el tratamiento son muy diferentes.

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Organization must come, in all states and in all sciences. The situation must be accepted; but in that situation, as in all political situations, there are great dangers as well as great opportunities. Organization is no substitute for genius. No amount of bureaucracy could have discovered penicillin.

—*Ogilvie*

Perianal Granular Cell Myoblastoma

Report of a Case

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A case of perinal granular cell myoblastoma is reported.

A search of the literature reveals a paucity of well-documented cases. Those available are collected and described.

GRANULAR cell myoblastoma was first described in 1931 by Abrikossoff.[1] Since then a large number of cases have been reported, the lesion is rarely mentioned as anal. A review of pertinent facts with regard to this curious tumor is presented, and a case is described.

Etiologic Background.—It is generally agreed that this tumor may arise anywhere in the body by dysontogenetic inclusion of primitive myoblasts.

Distribution.—In the majority of cases the tumor appears in the tongue. It also appears in decreasing frequency in the skin, subcutaneous tissue, muscle, maxilla, larynx and vocal cord, breast, mandible, lip, trachea and bronchi, ear, alveolar process and anus. Single cases have been reported of its occurrence in the urinary bladder, esophagus, lacrimal sac, floor of the mouth, pharynx, spermatic cord, urethra and vulva.

General Description.—These tumors are generally small (0.5 to 2 cm.) and sharply circumscribed. There may be encapsulation. Elevation of the skin is usual. The cut surface is finely nodular or lobulated and is yellowish to pearly white.

Microscopic Picture.—The microscopic description of this lesion has been well documented by Simon.[2] Characteristic large polyhedral or cylindric cells are present, containing abundant coarsely granular acidophilic cystoplasm. The nuclei are oval.

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The tumor cells are supported in a thin, poorly vascularized and somewhat delicate connective tissue septum that encloses groups of cells, giving at times an alveolar appearance. Malignant variants do occur. Another condition often associated with granular cell myoblastoma is proliferation of the overlying epithelium.

Seven or more true cases of overlying squamous cell carcinoma have been reported. In the absence of metastatic lesions from overlying epidermoid carcinoma, an exact differentiation between pseudoepitheliomatous hyperplasia and true overlying squamous carcinoma is difficult to make.

Abrikossoff[3] originally described four varieties of muscle tumor:

Type I: Tumor composed of myoblasts, completely devoid of striations.

Type II: Tumor similar to Type I, but with rare longitudinal or cross striations, or both, imperfectly developed in the margins of the cells.

Type III: Tumor with markedly hypertrophic myoblasts measuring 40 to 100 microns, often multinuclear and syncytial.

Striations may be seen in some of the cells, especially in the peripheral cytoplasmic zone, but are absent from many.

Type IV: Atypical myoblastic sarcoma, the cells of which show pleomorphism, some zones appearing sarcomatous and others showing more highly differentiated cells with well defined longitudinal and cross striations.

Most reviewers agree that Type IV is more properly classified as true rhabdomyosarcoma.

Malignant transformation has been observed in cases of Types I, II, and III, up to 2 per cent. The tumor may invade or metastasize.

Histogenesis.—Rothschild,[1] in a brilliant article discussed the histogenesis of this tumor. An outline of the various possible sources is presented.

1. *Myogenic:* The myogenic possibilities are as follows:

a. From myoblasts arising in muscle after trauma or inflammation.
b. From myoblasts in misplaced embryonic rests.
c. From myoblasts[3] arising in totipotent mesenchymal cells in the vicinity of small blood vessels.
d. From myoblasts arising from active muscle regeneration, representing only a hyperplastic healing process and not a true neoplasm.
e. From breakdown of skeletal muscle cells.

2. *Storage Cell Theory.* According to this postulate, these tumors represent mesenchymal cells or histiocytes of unusually large size, containing products of cellular metabolism and thus arising from muscle fibers or glandular epithelium which is undergoing necrobiotic change.

3. *Neurogenic Theory:* There is also a theory that the tumors represent altered fibroblasts of neural origin.[4]

Review of Literature.—A search of the literature reveals a few well-documented reports of this tumor as it occurs in the anorectal area.

Szathmary[5] in 1937 reported on a perineal tumor in a 32-year-old woman. It had existed for several months and was locally excised.

Crane and Trembley[6] in 1945 mentioned a walnut-sized perianal tumor. No other details were given.

Simon[2] reported the case of a 50-year-old woman who suffered from hemorrhoids for twenty years. A "seed" developed alongside the anus, grew and became painful. Examination revealed a nodule the size of a filbert, resembling a thrombosed hemorrhoid. The gross description is as follows:

"In the largest hemorrhoidal tag immediately underneath the epidermis, a sharply circumscribed, but non-encapsulated nodule 1.2 cm. in diameter, spherical in shape, was seen. It was firm and on cut section presented a yellowish-grey homogeneous surface in which no lobulation or granularity was noted."

London[7] reported the case of a 41-year-old man with urinary symptoms. Rectal examination revealed a spheroid mass on the left side of the rectum, above the prostate gland. A perineal prostatectomy was performed, with removal of a multilobular encapsulated tumor. The pathologist diagnosed this as a myoma. It is presented here because it is in the same family of tumors.

Murphy, Dockerty and Broders[8] searched the literature up to 1949 and collected 229 cases of granular cell myoblastoma, in which 5 of the tumors occurred in the buttock and 2 were of anal origin. The latter two cases were not described.

Rothchild and Crary[16] reported 2 cases in 1953.

1. A 36-year-old Negro was subjected to hemorrhoidectomy. On routine pathologic examination of the speci-

men a small granular cell myoblastoma was found in a skin tag. No unusual symptoms had been present, and the patient made a full recovery.

2. A 28-year-old white man presented a slowly growing firm and painless tumor in the subcutaneous tissue overlying the right gluteus maximus muscle. It had been present for two years. The lesion was excised locally, and the wound healed *per primam*. On cut section the specimen presented a circumscribed yellow area with a homogeneous surface. The patient was lost to follow-up study.

Arthur Purdy Stout,[9] in a personal communication, mentioned 5 cases with sketchy records.

1. A 40-year-old woman had a tumor 1 cm. in diameter in the perianal skin. Pseudoepitheliomatous hyperplasia was present.
2. A 30-year-old man had a tumor 2 cm. in diameter in the perianal skin. Again, pseudoepithliomatous hyperplasia was present.
3. A 30-year-old Negro had a small tumor at the anal orifice.

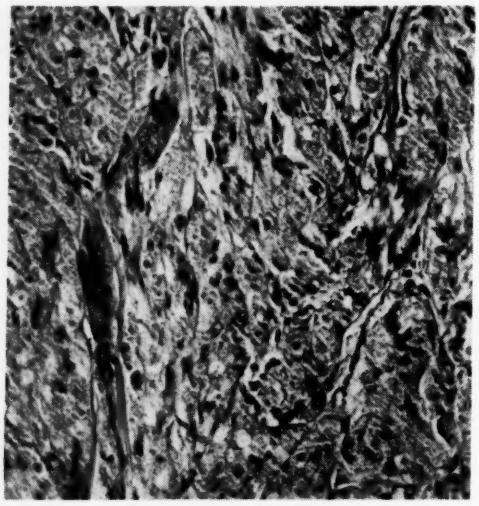

Fig. 1.—Microscopic appearance, × 10.

Fig. 2.—Microscopic appearance, × 440.

4. A 28-year-old man had a 5 mm. tumor in the mucosa and submucosa, 1 cm. inside the sphincter muscle.
5. An adult woman, age unknown, passed a small mass of tissue *per rectum*. It proved to be a granular cell myoblastoma, but the origin was never discovered.

Seven reports of tumors of the gluteus muscle were reported, but do not properly fall into the anorectal category. Several of these were malignant.

To the aforementioned cases a new experience is here added.

REPORT OF CASE

S. F., a 38-year-old white man, was first seen on April 12, 1958, complaining of an anal "growth" of four years' duration. There had been no pain or bleeding, but he had noticed a recent increase in the size of the mass. The previous history was completely normal, and the patient was in excellent health, compatible with his occupation of a teacher of physical education. Examination revealed a firm white walnut-sized tumor on the left side of the anal verge. It was not attached to the underlying or bodering structures.

The patient was admitted to the Rolling Hill Hospital, Elkins Park, Pennsylvania, and

operation was performed on April 23. Caudal anesthesia was employed. Sigmoidoscopic study to 25 cm., revealed no abnormality, and no other pathologic change was evident. The tumor was well circumscribed and easily removed. The wound was closed with No. 0 plain gut in layers. The fat of the ischiorectal space and the skin were also closed in layers. The patient made an uneventful recovery and has remained well.

A pathologic report made by Dr. Charles Harris follows:

"*Gross:* Specimen labeled tumor of buttocks consists of a large ellipse of skin measuring 3 cm. in diameter and 2 cm. in width, overlying 0.5 cm. of subcutaneous tissue. Centrally located is a large white slightly elevated area 1.5 cm. in diameter, elevated for a distance of 0.2 cm. above the general skin surface. The cut surface reveals an epidermal layer overlying a yellow-glazed mass, which grossly resembles anthoid tissue. The mass is clearly demarcated from the subjacent cuts. One section was taken.

"*Microscopic:* Sections of the lesion from the buttocks are covered by normal epidermis, with several retes extending deeply into the subjacent tissues. The rete pegs are generally sharp. The subjacent tissues are formed mainly by a nodule composed of large pink cells, with small nuclei often placed eccentrically. These resemble muscle fibers in some respects, but instead of showing the usual cross striations are granular. Sometimes the cells line up in parallel fashion to form small fascicles. The nuclei are often round, sometimes elongated and somewhat pyknotic. The lesion is not encapsulated but fairly well demarcated from the adjacent stroma.

"*Diagnosis:* Granular cell myoblastoma."

SCHLUSSFOLGERUNGEN

Es wird ueber einen Fall von perianalem aus granulierten Zellen bestehendem Myoblastom berichtet.

Die Durchsicht der Literatur ergibt nur wenige klar bewiesene Faelle dieser Art. Diese werden zusammengestellt und beschrieben.

RIASSUNTO

Viene riferito in caso di mioblastoma perianale a cellule granulose. Una rassegna della letteratura dimostra che i casi di questo tipo, ben documentati, sono molto scarsi. Essi sono stati reccolti e studiati.

CONCLUSIONS

Un cas de myoblastome à cellules granulomateuses de la région péri-anale est décrit. La littérature ne révéle que peu de cas bien documentés, qui sont ici analysés et commentés.

CONCLUSIONES

Se trata de un caso de mioblastoma granular perianal. Una revisión de la literatura ha demostrado una gran escasez de casos biem documentados.

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Editorial

Notes on the Role of Physicians in the Military Service of the United States

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THE term "military service" is here used to denote the United States Army and does not include the many important advances in medicine and public health developed by our sister services in the Armed Forces.

During the past one hundred and eighty-four years the Medical Department of the Army has had reason to be proud of its growth and achievements. Its history is replete with the names of physicians who have rendered service, either as medical administrators, specialists and research scientists or in other fields of endeavor pertaining to medicine.

Before discussing the specific roles played in the purely medical field, I should like to say something about physicians who have held important positions in the Army as administrators and leaders outside of medicine.

When I entered the military service, both the Chief of Staff and the Adjutant General were physicians. They were the two most powerful Officers in the War Department. The general staff at that time had not been developed to the extent it was to be at a later date, and the Army Service Force was still unborn. Both of these men were of strong character, able and efficient. That they did not get along too well with each other would be stating it mildly.

The Chief of Staff, General Leonard Wood, entered the Medical Corps one year after his graduation from Harvard Medical School. In 1898 he was authorized to raise the First Volunteer Cavalry Regiment (Rough Riders). Theodore Roosevelt was his Lieutenant Colonel. On July 8, 1898, he was promoted to the rank of Brigadier General of Volunteers and six months later to that of Major General. In February 1901 he was mustered out and appointed Brigadier General in the regular Army. He was appointed Chief of Staff in 1910.

Fred C. Ainsworth, the Adjutant General, was eight years older than General Wood and entered the Medical Corps in 1874. He served there until 1899, when he was made Chief of the Pension and Record Office. In this capacity he did a monumental job in compiling records and published "Official Records of the Union and Confederate Armies." He became Military Secretary of the Army in 1904 and was made Adjutant General in 1907.

If we go back into the history of the Army in the early years of the republic, we find some highly interesting events in which physicians played important roles. In the Revolution many physicians participated, some in their professional capacity and others as members of line organizations. After the Revolution, we had no Army to speak of for many years. On Jan. 3, 1784 Knox, the Secretary of War, reported that the Army was down to 700 "rank and file." Later on a further re-

duction took place, leaving only 25 men at Fort Pitt and 55 at West Point. Congress held that a standing Army in peacetime was "inconsistent with the principles of republican governments, dangerous to the liberties of a free people."

Our people were pushing westward and were having trouble with the Indians, who resented being routed out of what they considered their domain. The settlers in Ohio, Western Pennsylvania and Kentucky raised their voices to such an extent that something had to be done.

In 1789 Arthur St. Clair was appointed governor of the "Northwest Territory." He was also a Major General in the Army. St. Clair was born in Scotland in 1736, the grandson of the Earl of Roslyn, and studied medicine in Edinburgh prior to 1757, when he became an ensign in the British Army. He made an excellent record in the French and Indian War and, as a result, was given a large grant of land in the Ligonier Valley of Pennsylvania, which made him the largest land owner west of the Alleghenies. When the Revolution started, he threw his lot in with the colonies. His military career went up and down in this conflict, but after the war, evidently, he regained some of his prestige. On Nov. 4, 1791, he fought a battle with the Miami Indians under Little Turtle at St. Marys, Ohio. His troops were untrained, and he suffered a defeat that cost the lives of a very large part of his force. He continued to serve as governor until 1802, when he was relieved to live in poverty until Congress refunded the money he had spent out of his own pocket for pay and supply of his troops.

Another physician of this era who was active in military and political affairs was James McHenry, for whom Fort McHenry just outside Baltimore was named.

McHenry was born in Ireland in 1753. His family moved to America, and he studied medicine under Dr. Benjamin Rush of Philadelphia. He entered the Army as a Medical Officer and was taken prisoner by the British. Shortly after his release in 1778, he became General Washington's Secretary. After the War he served in the Maryland Senate and as a delegate to the Constitutional Convention. He was a signer of the Constitution and was appointed Secretary of War in 1796. When Mr. Adams was elected President, he retained him for one year but had to demand his resignation, as McHenry was an ardent Federalist and was alleged to be working against the President, politically.

During the months of 1960 and as long thereafter as possible, the editorial pages of the Journal of the International College of Surgeons *will be honored by presentations of rare and extraordinary historic value, the lectures presented at the International Surgeons' Hall of Fame on medical and surgical history from the earliest recorded achievements to those of our own times. We believe that* Journal *readers who were not able to attend the lectures will find them as exciting as did the audiences present at their delivery. The story of progress in the healing arts and the giants of medicine and surgery to whom we owe the amazing brilliance and scope of modern achievements is intrinsically thrilling and is a vital part of the education of every cultivated man and woman, inside or outside the profession. It is altogether fitting that it should appear in these pages, since the International College of Surgeons is first, last and always a teaching institution.*

When the War of 1812 started Madison was President, and the Secretary of War in his Cabinet was William Eustis, a phisician, who was born in 1753. At the outbreak of the Revolution he was practicing medicine in Boston. He entered the Army as a medical officer and served throughout the War. After the Revolution he served in Congress from 1801 to 1805. He was appointed Secretary of War in 1809, serving until 1813. (He also returned to Congress from 1819 to 1823.)

Next under the Secretary of War were the two Major Generals of the Army. The senior of the two was Henry Dearborn, a physician who had left his practice in Portsmouth, New Hampshire to enter the Continental Army. After the Revolution he was twice elected to Congress and served in Jefferson's Cabinet as Secretary of War for eight years. On Jan. 27, 1812, he was appointed Senior Major General of the Army. In 1815 he resigned to become Minister to Portugal.

The other Major General of the Army was General Pinckney, who was then 63 years of age. He was not a physician and had had no connection or association with the Army since the Revolution.

At that time the Army had nine Brigadier Generals; the senior, James Wilkinson, was a physician.

Wilkinson was born in Maryland in 1757 and studied medicine in Philadelphia. He entered the Army at the outbreak of the Revolution and became a close friend of Aaron Burr and Benedict Arnold. In the Saratoga Campaign he is alleged to have appropriated as his own certain information as to the strength and position of the British, which had been secured by Colonel John Hardin. He was a protegé of General Gates and in 1778 was appointed Secretary of the Board of War. He was implicated in the Conway Cabal and resigned his brevet Brigadier General Commission but kept on for a time as a colonel. In 1786 he founded Frankfort, Kentucky, and entered into a deal with Governor Esteban Rodriguez Miro to bring Kentucky under the protection of Spain. For this he drew an annual "pension" from Spain. Notwithstanding all this, he was commissioned a Lieutenant Colonel in 1791 and served under General Anthony Wayne against the Indians in Ohio. When Wayne died, Wilkinson was made Commander in Chief of the Army. He was investigated many times but managed to escape punishment.

Jacobs, in his book *The Beginning of the United States Army*, is justly critical of the general officers of the line. He has the following to say about the staff:

"The so-called 'General Staff' was no better than the field command, probably knowing even less of its characteristic duties. Thomas H. Cushing, the Adjutant General, was in such poor health that he could scarcely get about. The Inspector General, Alexander Smyth, was just a commonplace braggart without redeeming qualities. Morgan Lewis was Quartermaster. Although he was a person of character, he had only a microscopic conception of the wide range and extreme difficulties of his task. The Adjutant and Inspector, A. Y. Nicoll, had the rank only of a Major and his contribution to the Service was in proportion. Robert Brent, the Paymaster, possessed a knowledge of peacetime routine, but that was about all. As a rule, the 'General Staff' functioned as individuals and not as a team."

Is it not a wonder that we came out of that War as well as we did?

Now let us go back in history to the time of the Revolution and consider some of the things that pertain more to medicine *per se* than those I have thus for mentioned.

When the American Revolution started, there was no medical service except that given when and where fighting took place,

by those, qualified or not, who happened to be there. After George Washington was appointed in mid-June of 1775, he realized that there was need for some form of medical service, and on July 27 Congress authorized the establishment of "an hospital." Many physicians were active in the Revolution; one of the most famous was Benjamin Rush of Philadelphia. After the War he wrote the first American textbook on psychiatry, in which he advocated humane care and treatment of the mentally deranged.

Dr. Rush probably enjoys today the greatest medical reputation of all the physicians of our Revolutionary period. Ashburn, in his *History of the Medical Department*, says in a footnote:

"By virtue of his social and professional prominence, his position as teacher and his facile pen, Benjamin Rush had more influence upon American medicine and was more potent in the propagation and long perpetuation of medical errors than any man of his day. To him, more than to any other man in America, was due the great vogue of vomits, purging, and especially of bleeding, salivation, and blistering, which blackened the record of medicine and afflicted the sick almost to the time of the Civil War."

In 1800 Dr. Benjamin Waterhouse, a surgeon of the Army, introduced vaccination for smallpox into the United States and was such a crusader for its adoption that he was referred to as the Jenner of America. The first person he vaccinated was his own 7-year-old son; then followed his three other children. He then exposed all of them to smallpox, and none of them contracted the disease.

Dr. William Brown, who replaced Dr. Rush as physician to the Middle Department, wrote *Pharmacopia for the Use of Army Hospitals*. This was the first American pharmacopia. A copy of it is in the Toner Collection in the Library of Congress.

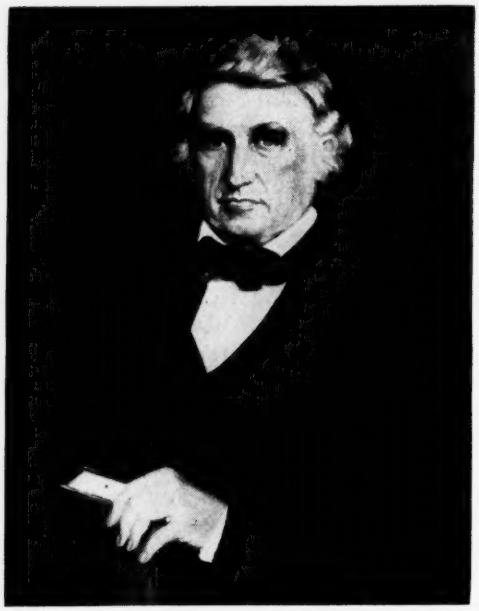
The War of 1812 found the Medical Department without a head; Congress in 1813 therefore authorized the appointment of a Physician and Surgeon General and an Apothecary General.

Dr. Tilton of Delaware was appointed to the former position, mainly, it has been claimed, because he had published a pamphlet entitled *Economical Observations on Hospitals* and dedicated it to General John Armstrong, Secretary of War.

Ashburn, in his History of the Medical Department, makes the following comment on this literary effort:

"This pamphlet consisted of three parts, the first a wholesale condemnation of general hospitals and criticism of the existing organization of the department; the second largely a dissertation on camp infection (pollution) and its avoidance; and the third a description of his own plan for a hospital. 'In such cases (cold climates and winter seasons) the best hospital I have ever contrived was upon the plan of an Indian hut. The fire was built in the midst of the ward, without any chimney, and the smoke circulating in the midst of the ward, passed off through an opening four inches wide in the ridge of the roof. The common surface of the earth served for the floor. The patients laid with their heads to the wall round about, and their feet were all turned to the fire. The wards were thus completely ventilated. The smoke contributed to combat infection, without giving the least offense to the patients; for it always rose above, before it spread abroad in the ward. And more patients could be crowded with impunity in such wards, than in any others I have seen tried. This was the expedient I employed in the hard winter of '79, '80, when the Army was hutted near Morris Town, and I was well satisfied with the experiment.'"

One of the best known physiologic experiments was the study of digestion by William Beaumont, who entered the Army



Dr. William Beaumont.

as a Surgeon's Mate in December 1812, resigned in 1815 and was reappointed in 1819. On June 6, 1822, a French Canadian named Alexis St. Martin received an accidental shot gun discharge in his left breast at a distance of only two feet. Stomach and lungs were both exposed, and the former organ was perforated, the opening being covered by a flap of skin. Beaumont treated him in the hospital for ten months, after which the wound was partly healed and the civil authorities ordered him sent home—a distance of 1,500 miles. Beaumont knew that this would cause his death, so he kept him in his home, clothing, nursing and feeding him at his own expense, including that of transporting him to a new station in 1825. All this time Beaumont was carrying on studies of gastric digestion. His first report was presented before the Michigan Medical Society in 1827.

Beaumont's studies continued for some time, and in 1833 he published the results of 240 experiments in a book entitled *Experiments and Observations of the Gastric Juice and the Physiology of Digestion,* a monumental study that still constitutes the background of all gastric physiology.

In 1834 Beaumont was ordered to Jefferson Barracks, Missouri, and Lewis Cass, the Secretary of War, was petitioned by a large group of members of Congress to send him and St. Martin to Boston for further study in conjunction with Dr. Charles Jackson. Cass stated that this would be impossible.

Baron Larrey, Napoleon's chief surgeon, had been an advocate of treating the wounded during and not after a battle. Until the Civil War no provision had ever been made for removing the wounded from the battlefields. In fact, after some of the earlier battles of that war the relatives and friends of the wounded would search the fields and if they were so fortunate as to find them, would take them away in carriages. Attempts were made on several occasions to establish an ambulance service, but they always failed because of ignorance, apathy or plain stupidity. Owing to the excellent work of a Sanitary Commission appointed by Secretary of War Cameron, a bill was put through Congress reorganizing the Medical Department and appointing a 34-year-old officer (Hammond) as Surgeon General. Hammond had served twelve years in the Army and had always been active and studious. In 1857 he had been awarded the American Medical Association Prize for an essay, "The Nutritive Value and Physiological Effects of Albumen, Starch and Gum When Singly and Exclusively Used as Foods."

Hammond appointed Jonathan Letterman as Chief Surgeon of the Army of the Potomac, and Letterman introduced system, order and efficiency into the evacuation of the wounded. His system is the

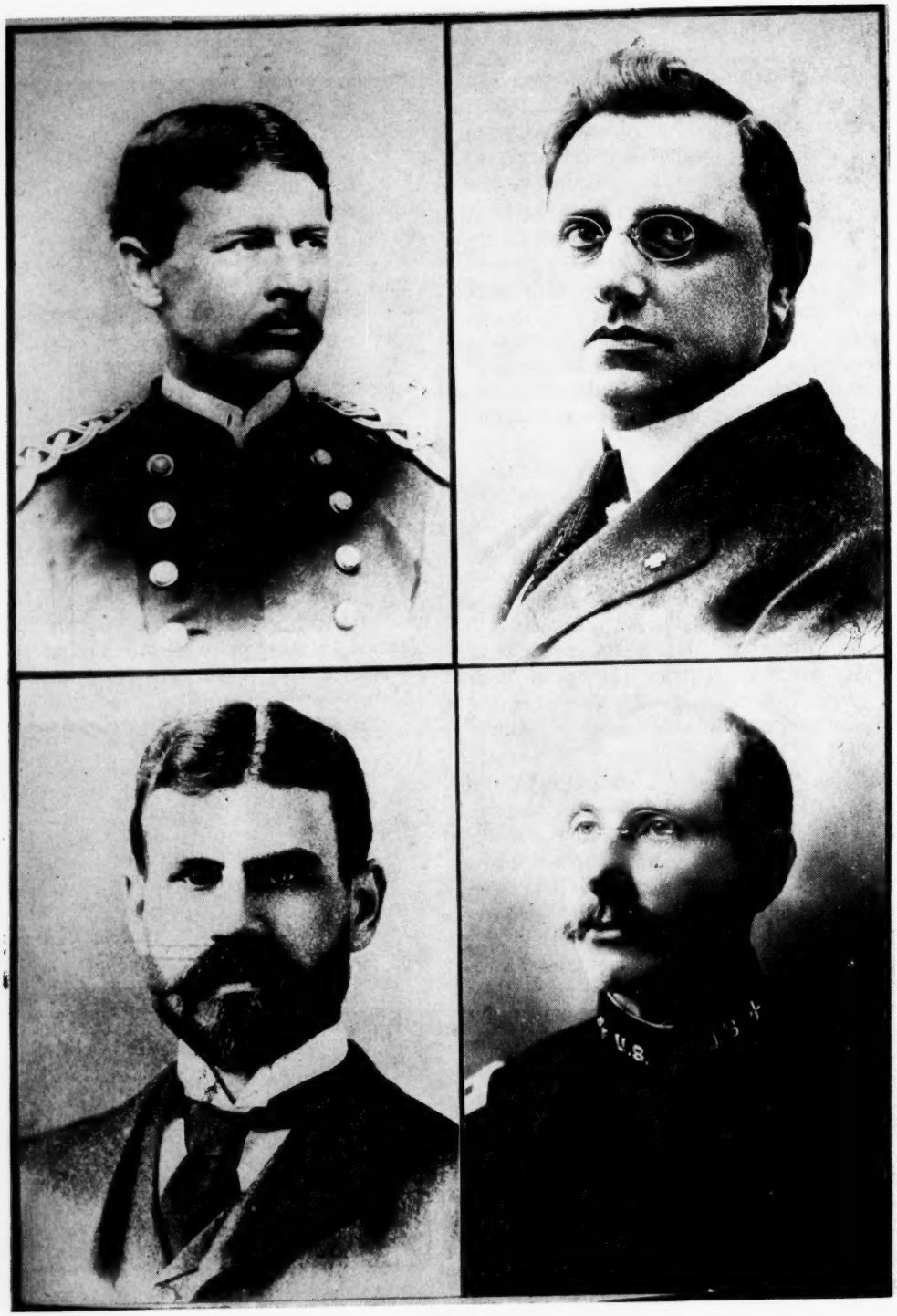
basis of medical field service now used in most of the armies of the world today. This was a wonderful advance, as even at Bull Run (Aug. 29-30, 1862) many of the wounded of Pope's Army lay on the field for a week.

In 1863 Surgeon General Hammond appointed Dr. John H. Brinton a Surgeon of Volunteers from Philadelphia to arrange for a school for medical officers in Washington. Lecture rooms and a laboratory were set up in the Corcoran Building. It was only necessary for Mr. Stanton, the Secretary of War, to approve and authorize the ordering in of teachers and students. On his way to the office one morning, his carriage stopped at the building and Stanton walked hastily through the rooms. His only remark was "Ugh." When he arrived at his office he sent for the Acting Surgeon General and said, "Are these lectures to be given in the evening?" To an affirmative reply, he growled, "They will go to the theatre and neglect their duties. It shan't be." That was the end of the Army Medical School for thirty years.

At the outbreak of the Civil War the Surgeon General's library consisted of about 1,000 volumes. Dr. John Shaw Billings, who had been a distinguished medical officer in the Civil War, had been detailed in 1869 to the Treasury Department for the purpose of inspecting and improving the Marine Hospital service, the predecessor of the present Public Health Service. It had not been developed and was not in very good shape when Billings took over. He formulated plans for the reorganization, which were followed and, which marked the beginning of the growth of this service. He served as consultant to the Public Health Service until 1874. He was then given the task of building up the Surgeon General's library.

Billings was a man of vision, possessed of great energy and perseverance. In 1879 he published a "specimen fasciculus" of a combined index catalog, for one letter only, of authors and subjects arranged in dictionary order, and in 1880 he issued the first volume of the indexed catalog. Dr. Billings also had charge of the vital statistics of the United States Census. According to the London Times, as cited by Ashburn in his history of the medical department, the census records prior to 1880 were valueless. Billings had charge of the tenth, eleventh and twelfth censuses, as a volunteer worker, and from a state of chaos he brought the vital statistics of the United States up to a most satisfactory condition. In 1875 he began to plan the Johns Hopkins Hospital. His plans included not only buildings but organization and administration, particularly including the teaching of medicine and surgery. As he himself said, he had planned to equal or excel any similar institution in the world. The Hospital was

Albert J. Myer.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opened in 1889, and it fulfilled the plans he had made for it. He was also instrumental in gathering the first group of men who made that institution world famous.

When Dr. Billings retired from the Army in 1895, he went first to the University of Pennsylvania. A year later, however, he was chosen to organize the New York City Public Library, then consisting of three separate institutions. He first made a trip to Europe and began the work of classification and cataloging. A new building was erected according to his sketched plans, and was opened in 1911.

Many attempts have been made to secure proper housing for the Surgeon General's Library. I recall a large framed architect's drawing, prepared at the end of World War I, that hung in the office of the Surgeon General. Numerous bills were introduced into Congress but failed of passage. In the middle of the '30s, a bill was given hearings before the Committee on Military Affairs of the House of Representatives, of which Mr. May of Kentucky was Chairman. The Surgeon General was told that there was no reason why the bill should not be reported out of Committee favorably. Toward the end of the hearings, Mr. May was suddenly called to The White House in the late morning. Immediately after lunch he telephoned the Surgeon General and said that he had been ordered to "kill the bill." This, of course, was a great blow.

During the War the incunabula of the library were sent to Cleveland, Ohio, for safekeeping, and there they remained. The library, however, now known as the National Medical Library, is on its way to be housed in a suitable building on the grounds of the National Institute of Health in Bethesda, Maryland.

The Signal Corps of the Army originated from the hobby of a medical officer. Assistant Surgeon Albert J. Myer, the man for whom Fort Myer, Virginia, is named, was interested in mechanical and manual signaling. He started by studying the smoke signals used by the Indians. In June 1860 he was given a commission as Major and Signal Officer, the only one in the Army. He proceeded to organize the Signal Corps and was made Chief of that Corps, with the rank of Brigadier General, in 1880.

In 1819 the Surgeon General of the Army, instituted the recording of weather observations by medical officers at all posts. The first observations were of temperature only. Later were added the measurement of rainfall and, in 1843, wind direction and notes as to sunshine, cloudiness, rain or snow.

In 1844 Surgeon General Lawson had these diaries abstracted and tables of mean temperature prepared. The meteorologist of the War Department, Mr. James P. Epsy, worked in the Surgeon General's office and in 1841 published *Philosophy of Storms*. He had devised a series of skeleton maps on which he could show, by appropriate symbols, the direction and force of the winds and the barometric fluctuations at the points of observation, thus predicting the shape and size of the storms, with their direction and the velocity of their motion. This was the forerunner of the weather bureau maps of today. Officers were then directed to record barometric observations, clearness of the sky, force of wind on decimal scales, direction and velocity of clouds, wet-bulb readings, and other noteworthy phenomena. By 1860 these observations filled 64 large folio volumes in the office of the Surgeon General. In 1870 the work was

Opposite: The Reed Board, which in 1900-1901 solved the century-old problem of the course of yellow fever. Dr. Walter Reed (*above, left*); Dr. Aristides Agramonte (*above, right*); Dr. Jesse W. Lazear, who lost his life as a result of the experiments (*below, left*), and Dr. James Carroll (*below, right*).

taken over by the Signal Corps, but the observations continued as a part of the post surgeon's duties in many posts for some years after the Spanish-American War. In 1890 the Weather Bureau became a part of the Department of Agriculture.

Toward the close of the last century and in the early years of the present one, great practical advances were made in the study of infectious diseases. Medical Officers of the British and French Armies had contributed extremely valuable work in this field. The first American Army Surgeon to take up the study of bacteriology was George M. Sternberg (1839-1915). He isolated the pneumococcus in 1880, simultaneously with Pasteur. He published the first complete American text on bacteriology in 1896, as well as many valuable papers on bacteriology and disinfection. He also added to the profession's knowledge of yellow fever, as a negative observation, by isolation of Bacillus X. This Bacillus X was the organism described by Sanarelli and called, by him, Bacillus icteroides. It was Sternberg who, as Surgeon General, formed the Yellow Fever Commission headed by Major Walter Reed. Reed had studied under Professor Welch at Johns Hopkins and had done special work on the pathologic aspects of typhoid fever. Assisting Reed on the Board were Drs. James Carroll, Aristides Agramonte and Jesse W. Lazear.

Yellow fever was endemic in Havana, and epidemics of this dread disease occurred in many places on our shores, from the Gulf of Mexico to New England.

Walter Reed was born in Gloucester County, Virginia, and grew up there during the Civil War. He received his M.D. degree at the University of Virginia in 1869, at the age of 17. He then went to New York, obtained a second M.D. from Bellevue College Hospital and served an internship at the Brooklyn City Hospital and at Blackwell's Island. At 21 he was a district physician in New York City and at 22 one of the five inspectors of the New York City Board of Health. Reed entered the Medical Corps of the Army in 1875 and proved to be the ideal man to undertake the task of studying yellow fever.

The Commission arrived in Cuba in June 1900, and, as Reed knew local conditions, he established his laboratory six miles outside Havana. The Commission first instructed some volunteers to sleep in a screened room with the clothing and bedding of the yellow fever patients. None in this group were infected.

As early as 1881 Dr. Carlos Finlay, a Cuban physician, had advanced the theory that yellow fever was transmitted by a mosquito. He was scoffed at when he voiced this idea, but Reed set out to investigate it. Fourteen volunteers were bitten by mosquitoes that had bitten yellow fever patients three weeks earlier; into 6 the blood of patients with yellow fever was injected, and 2 were given injections of filtered blood serum from "positive" cases. In 22 cases yellow fever developed thus proving that the disease was mosquito-transmitted as well as that it was due to a filterable virus.

Dr. Carroll allowed a mosquito to bite him. As a result, the disease developed in a mild form, and he recovered. Dr. Lazear, while working with infected mosquitoes, noticed that one had settled on his arm, and he allowed it to feed. Yellow fever developed, and he died.

William C. Gorgas was the Chief Sanitary Officer in Havana at the time of the discovery of the mosquito transmission. He worked in close cooperation with Reed and began at once to eliminate breeding places, kill larvae and adult mosquitoes and protect human beings by screening houses, etc. As a result, Havana became free of the disease for the first time in a hundred and fifty years.

An interesting sidelight on Reed's work was cited by Dr. Walter Bierring in a lecture given in November 1957, at the Walter Reed Army Medical Center. His quotation was from a letter written by Reed to his wife on New Year's Eve, Dec. 31, 1900, and is as follows:

"Only 10 minutes of the old century remains; here I am sitting reading that most wonderful book 'LaRoche on Yellow Fever' written in 1853; 47 years later it has been permitted to me and my assistants to lift the impenetrable veil that has surrounded the causation of this most dreadful pest of humanity and to put it on a rational and scientific basis. I thank God that it has been accomplished during the latter days of the old Century; may its cure be wrought out in the early days of the new."

With this background of Havana experience, Gorgas was the logical man to appoint as Chief Health Officer of the Panama Canal. The Isthmus of Panama had been called "the white man's grave" not only on account of yellow fever but because many other infectious diseases occurred there and caused the very high morbidity and mortality rates. Gorgas knew exactly what he wanted to do; he was beset, however, by political and bureaucratic troubles that made his work extremely difficult at first. He was a persistent, quiet sort of man, not easily upset or angered. He also had the backing of the President of the United States. The actual work of the Canal was in charge of engineers, who understood very little about the prevention of disease. The program laid down by Gorgas was an expensive one, naturally, consisting of mosquito control, isolation of patients, proper medical care, and not only prevention of the breeding of mosquitoes but the elimination of breeding places as far as possible. The medical history of the building of the Panama Canal is a fascinating one, but cannot be gone into here.

General W. C. Gorgas.

When the Spanish American War came, our profession knew little or nothing about diseases occurring in the tropics. In fact, it was not a tropical disease that caused high morbidity and mortality rates among our troops during that war, but it was one well known to us, i.e., typhoid fever. This was the subject of an investigation by a commission set up by the President. It was several years later that successful prophylaxis against this disease was put into effect.

In 1908, the Surgeon General sent Major F. F. Russell to Europe to study the methods of typhoid prevention used in the British and German Armies. Major Russell returned to the United States, and the Surgeon General appointed a Board of distinguished civilian physicians to work with him. They recommended that a typhoid vaccine used by the British be given to our troops, Major Russell having brought back cultures of the strain of organisms used by the British in preparing the vaccine.

This vaccination of troops met with a certain amount of objection on the part

of the general staff. Nevertheless, an order was promulgated making vaccination compulsory in all persons under 45 years of age. As the reason for this arbitrary age limit, I was told that the Army General Staff of the Army was opposed to compulsory vaccination and that Russell and his co-workers suggested this age limit because nearly all members of the General Staff at that time were over 45. Apparently it "worked," but it had nothing to do with the epidemiologic aspects of the disease.

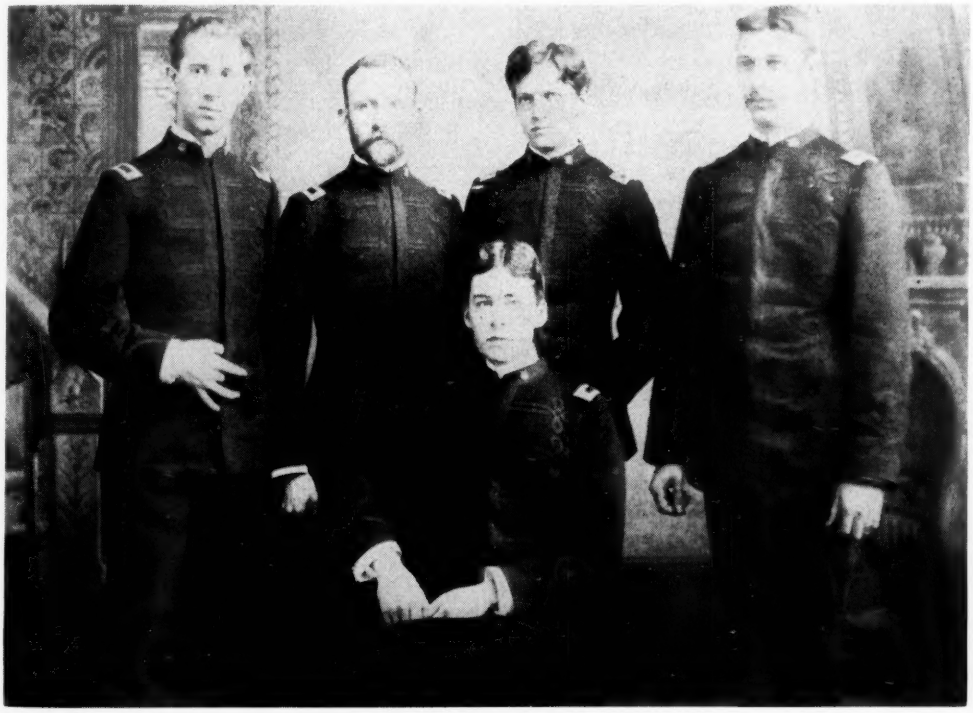
The fact that the disease was wiped out among our troops was due not only to prophylactic vaccination but to a number of other things—the purification of water supplies, the eradication of flies and the protection of foodstuffs. Nevertheless, the vaccination was a forward step and was one of the principal causes of elimination of the disease.

When our government first occupied Puerto Rico there was a widely prevalent disease known as Puerto Rico anemia or tropical chlorsis. It was soon determined that this disease was due to infestation with hookworm. Credit for investigation and control of this disease is due to Bailey K. Ashford, then a lieutenant, who had been taught helminthology at the Army Medical School by Prof. Charles Wardell Styles. By means of proper diagnosis and treatment, the disease was soon brought under control.

General Leonard Wood.

As our Army moved out to the Orient, it came into contact with a disease called beri-beri. This was an extremely prevalent disease in the Far East. As a matter of fact, during the Russo-Japanese War, the Japanese reported that on a given day nearly one quarter of the soldiers and sailors sick in hospitals were suffering from beri-beri. The statistics from this War are not complete, so only percentages are given. The Japanese really led the field in the investigation of beri-beri, as it had caused so much illness and death among their troops. During the winter of 1901 there were more than 2,000 patients with beri-beri in Bilibid prison in Manilla, and in the six months ending May 31, 1902, there were 2,470, with 77 deaths. A Board for the study of tropical diseases had been established in Manila by the Army, but it remained for Captain E. D. Vedder to make the study a personal and prolonged investigation. He was a member of the Board, but he was a meticulous investigator, and he "kept after it" for several years. Experiments with diet showed that persons who ate highly milled rice were the one who contracted beri-beri. This was due, as was proved later, to a lack of vitamin C, which is contained in the hull of the rice. Years later beri-beri was still prevalent in the Philippines, and the people knew that alcoholic extract of rice polishings, which they called tiki-tiki, would cure the disease. Through the work of the Tropical Board, in 1909 there were

General Leonard Wood (center) with friends.



First class graduated from the Army Medical School. *Left to right, standing:* Bratton, Quinton, Howard and Wilson; *seated,* Porter.

only 604 cases among the scouts and in 1911 only 2; they have been practically free from the disease ever since.

I would like to mention a few other medical officers without going into detail about their accomplishments.

B. J. D. Irwin, the first person to be awarded the coveted Medal of Honor for bravery, pioneered the treatment of wounded in tents at the battle of Shiloh (the tents were "borrowed").

Nicholas Senn of Chicago, a National Guard Medical Officer, founded the Association of Military Surgeons. He was responsible for many reforms in the medical service of the National Guard.

James C. Merrill was the author of a book entitled *Notes on the Ornithology of Southern Texas.*

Elliott Coes wrote *The Key to North American Birds.*

Carl R. Darnall was Professor of Chemistry and Adjutant at the Army Medical School when I entered the service. Darnall took an early interest in water purification in the field. He devised the Darnall filter, which was extensively used until it was displaced by chlorine purification of water. Darnall was a pioneer worker with chlorine in this connection.

I have tried to cover, as well as space permits, some of the outstanding events in which Army physicians have played leading roles. Many whose names have not been mentioned here have added to the sum of scientific and professional knowledge. The old reports sent in each year by medical officers in far places make interesting reading . These officers had little to work with and were far from medical centers as a rule; yet they carried on, and many have left their mark on our medical history. The era in which they lived is past. Transportation and communication have changed things so that the medical officer has close contact with his professional brothers in civil life and has modern surroundings and equipment with which to work. It is better so, but we should not forget those who led the way.

We do not know the cause of ulcers; when we do, operations will no longer be required for their cure. But we do know that they occur only in the presence of free hydrochloric acid; that they are common where acid is high, and unknown where it is congenitally absent or lost through disease; that in the normal alimentary canal they are confined to the segment exposed to the action of unneutralized acid, that is, between the cardia and the bile papilla, but that they may be found in any part of the intestine which, by operation, experimental anastomosis, or aberrant oxyntic tissue, is brought in contact with acid. The primary cause may be excessive acid production, appears to be due to the loss of this intrinsic factor; it may be cured by the administration of the essential principle in the form of liver extract, or of the intrinsic factor in preparations of gastric.

—*Ogilvie*

New Books

BOOKS RECEIVED

The following books have been received by the Editor; they will be reviewed critically as space and facilities permit. Omission of more extended review, however is not to be taken as criticism of the merit of the book.

Textbook of Surgery. Edited by Guy Blackburn and Rex Lawrie, with a foreword by Sir Russell Brock. Springfield, Illinois: Charles C Thomas, Publisher, 1958. Pp. 1122. Illustrated. *Reviewed in this issue.*

Le Diagnostic du Cancer d'Estomac a la Periode Utile (Diagnosis of Carcinoma of the Stomach at the Time Most Favorable for Treatment). By Rene A. Gutmann. Paris: G. Doin et Cie, 1956. Pp. 257.

Operative Surgery. Edited by Charles Rob and Rodney Smith. London: Butterworth & Co., Ltd.; Philadelphia, F. A. Davis Co., 1956. Vols. 1 and 2 of 8 (plus index). Profusely illustrated.

Le risque operatoire en chirurgie biliopancreatique (The Operative Risk in Biliary Surgery). By Yves Salembier. Paris: Masson et Cie, 1959. Pp. 260, with 2 illustrations.

The Triumph of Surgery. By Jürgen Thorwald. Translated from the German by Richard and Clara Winston. New York: Pantheon Books, Inc., 1959. Pp. 454, with 13 illustrations. *Reviewed in this issue.*

Functional Bracing of the Upper Extremities. By Miles H. Anderson. Springfield, Ill.: Charles C Thomas, Publisher, 1958. Pp. 463. Illustrated.

The Clinical Management of Varicose Veins. By David Woolfolk Barrow. New York: Paul B. Hoeber, 1957. Pp. 167, with 70 illustrations.

Pathology and Surgery of the Veins of the Lower Limbs. By Harold Dodd and Frank Cockett. Baltimore: The Williams & Wilkins Company, 1957. Pp. 462, illustrated.

Tumors and Tumorous Conditions of the Bones and Joints. By Henry L. Jaffe. Philadelphia: Lea & Febiger, 1958. Pp. 629, with 701 illustrations. *Reviewed in this issue.*

Pain: The Destroyer and the Sympathetic Origin of Dystrophy. By Miguel Lopez Esnaurrizar. Paris: Editions Lamarie, 1959. Pp. 210, with 18 illustrations. *Reviewed in this issue.*

Spinal Cord Compression. By I. M. Tarlov. Springfield, Ill.: Charles C Thomas, Publisher, 1957. Pp. 147, with 41 illustrations.

The Surgeon's Tale: The Story of Modern Surgery. By Robert G. Richardson. New York: Charles Scribner's Sons, 1959. Pp. 227, with 45 illustrations.

History of American Medicine. Edited by Felix Marti-Ibanez. New York: MD Publications (MD International Symposia), 1959. Pp. 181.

The Surgical Treatment of Facial Injuries. By Varaztad Hovhannes Kazanjian and John Marquis Converse. Baltimore: The Williams and Wilkins Company, 1959.

Traumatic Injuries of the Hand. By Enrique Carlos A. Defilippi Novoa. Buenos Aires (Argentina): Editorial Alfa, 1958. Pp. 313, with 139 illustrations.

Treatment of injuries of the hand, the exceptional importance of which is recognized, requires for efficient management the complete rules which are enumerated in this book, together with their explanations.

The surgeon should remember, especially in operation on the hand, that he works on an organ of great functional significance and that any error may result in permanent injury. The anatomic delicacy and complexity of the hand tends to complicate wounds and traumatic injuries, and it is a paradox of such injuries that, though slight in extent, they may affect a great number of structures and produce graver results than are readily apparent. It is therefore important that the emergency or general surgeon have the proper concepts and directions to permit the immediate treatment to solve the acute problem and still make easy the later repairs by the specialist.

The problem of open and closed trauma of the hand is secondary to the proper treatment of the lesions, and dependent on it. This book should be read by all general physicians as well as by surgeons.

—JUAN OLIVELLA, M.D.

Textbook of Surgery. Edited by Guy Blackburn and Rex Lawrie, with a foreword by Russell Block. Springfield, Ill.: Charles C Thomas, Publisher, 1958. Pp. 1122, illustrated.

Published first in London and Toronto, this monumental work has as its contributors a galaxy of names bright in the scientific firmament. We find chapters by H. J. B. Atkins, J. S. Batchelor, Guy Blackburn, R. H. F. Brain, Patrick Clarkson, J. D. Dow, N. L. Eckhoff, Rex Lawrie, W. S. McConnell, W. H. H. Merivale, Sir Heneage Ogilvie, A. G. Parks, D. N. Ross, Peter Schurr, T. T. Stamm, and A. B. Wayte, all of the staff of Guy's Hospital, London, but many with numerous other appointments.

The text, thorough and authoritative, was prepared specifically for the undergraduate student. Its emphasis is therefore on general surgery, such specialties as ophthalmology and oto-rhino-laryngology being omitted. The clinical aspects of surgery, with particular stress upon diagnosis, are emphasized.

Because of the original intent of the editors and contributors, references to the literature are also omitted, the purpose being to inform the student of the most modern procedures rather than to confound him with names.

The book runs the whole gamut of general surgery, clearly demonstrated in readable language and profuse illustrations, to prepare the student for his final examinations, as the editors point out, "without cramming." This is a refreshing approach to medical education, for which the writers saw, from experience, a definite need. It is hoped that the text will attract wide attention in the United States.

Beyond the group for which it was written, however, the book should be important to all medical school directors and those who teach.

—MAX THOREK, M.D.†

Mitral Valvulotomy. By Harold J. Stewart and Frank Glenn. Springfield, Ill.: Charles C Thomas, Publisher, 1959. Pp. 218, with 25 illustrations.

This treatise comes from the New York Hospital. It is an analysis of 300 patients who were operated upon for mitral stenosis. The analysis is complete and deals with every aspect of the subject. It is critical and factual. The book is highly recommended as a complete and detailed presentation of this subject. The introduction of the operation for mitral stenosis opened a new world to the authors, as it did to others, and made it possible for them to record their experience with it.

This is primarily the work of the medical cardiologist. The surgeon and his operations are in a secondary position. This relation is

†Deceased.

indicated by the following quotations: "Those who embark upon this work need the personnel facilities of a general hospital of some size. It is not a task to be undertaken singlehanded by a surgical group, able as they may be. The cardiologist and the internist, by tradition, training, and experience are better equipped in particular to recognize the conditions that are of great importance from a prognostic viewpoint. Furthermore their appraisal of previous therapy and trial medical management during evaluation for mitral valvulotomy are significant factors in predicting results. The supportive measures that the heart requires following the burden of operation the surgeon must learn from the cardiologist." "The selection of patients for mitral valvulotomy is best done by an able and a cooperative group of physicians who bring together all the relevant information that is possible to obtain. . . . The administration of anesthesia is an integral part of the operative procedure. . . . The surgeon's objective is to open the stenosed valve as adequately as possible so that the area of the valve orifice approaches that which would be normal for the patient. . . . A great deal has been learned in this period. Results are encouraging. . . . The total effect of valvulotomy must be determined by trying to ascertain whether or not it has relieved symptoms, overcome disability and prolonged life, as compared to the course of those patients not treated by this procedure."

Obvious as these statements are, it would appear that the surgeon should be informed of them by the internist who has managed therapy in the past, and surgical operation contributed by surgeons is merely incidental to management. No doubt the internist regrets that he does not operate.

The character of the analysis and the style of expression are indicated in the following excerpts: "The improvement rate was greater in the second and third hundred patients than in the first 100 patients not only in patients in whom the valve was estimated at operation to be tight mitral stenosis but also in those in whom the valve was estimated as moderate stenosis. The improvement rate was greater both in patients with marked calcification and also in those with no calcification, in the second and third hundred patients than in the first 100 patients but was approximately the same in the two series in those with moderate or slight calcification. The percentage of patients obtaining improvement with respect to their presenting symptoms was greater in the second and third hundred patients than in the first 100 patients. In both series an adequate split in a tight valve resulted in a high percentage of improvement (80 per cent, 88.9 per cent, respectively). In the first series, 11 patients with tight valves did not have good splits and only five of these 11 were improved. In the second series, only one patient with a tight valve did not have an adequate split and the status of this patient was unchanged by operation. In both series, approximately 70 per cent of patients with moderate stenosis and in whom good splits were secured was improved. In the first series, three of four patients having moderate splits, were improved, but none in this category was improved in the second series. In both series, there was no improvement in patients who had little or no stenosis even though some increase in the size of the valve was achieved." The results improved with experience, as has occurred in other clinics.

The authors are to be congratulated for their careful and exhaustive analysis of this complex and complicated subject. It is a high example of clinical investigation.

CLAUDE S. BECK, M.D.

Hypnosis in Anesthesiology. By Milton J. Marmer, with a foreword by John B. Dillon. Springfield, Ill.: Charles C Thomas, Publisher, 1959. Pp. 150.

For a long time there has been a real need for a textbook of this type. Although no one would write a textbook on a subject in which he had no interest or enthusiasm, those who know Doctor Marmer know that, although he is enthusiastic in this field, he brings to his work a high degree of conservatism and a broad intelligent approach. His textbook, therefore, is one that should interest anesthesiologists, persons interested in hypnosis and its application in medicine, and obstetricians as well. In fact, it is this reviewer's opinion that all members of the medical profession would do well to read and know more about hypnosis

because, if even the mildest form of suggestion is used, no man can escape influencing his patients by this suggestion or by the use of the spoken word. Even though one has a distaste for practicing overt hypnosis, the reviewer is sincerely convinced that an adequate course in hypnosis should be a prerequisite for every practitioner of the healing art. To those who have not had this advantage, Doctor Marmer's book represents a good substitute.

The text is well written, legibly and neatly printed and well organized, with few errors. There is, in addition, a thoroughly adequate index. It is organized into historical background, the psychophysiologic approach to hypnosis, the psychologic aspect of anesthesiology, methods of induction, and the use of hypnosis in anesthesiology, in pediatric anesthesia, in the therapy of pain states, in posthypnotic suggestion, in hyposemantic difficulties and precautions. Finally, the data are organized into a bibliography and index.

The reviewer of the text could easily get the feeling that Doctor Marmer was trying to tell someone what and how to do in the field of hypnosis. This I think he accomplished very well. Personally, however, I should have desired for the usual student a little bit more background in the psychophysiologic aspects of hypnosis and a little more on the medicolegal implications. Nevertheless the book is so well written that one finds oneself able to read it almost as though it were a novel rather than a textbook.

The book should be a "must" in all libraries used by anesthesiologists, nurses, medical students, surgeons and obstetricians.

—MAX S. SADOVE, M.D.

Surgical Service Guide. By Louis T. Palumbo. Chicago: The Year Book Publishers, 1959. 2d ed. Pp. 208, with 22 illustrations.

This valuable handbook was prepared for use in the postgraduate residency training program at the Veterans Administration Center in Des Moines, Iowa, of which the author is chief surgeon. After thirteen years of practical use, the text has now been revised and enlarged in a second edition, with the help of Dr. Palumbo's staff members. In his introduction the author states: "The surgical operation is only one phase in the treatment of a disease." He therefore begins with anesthesia and carries the reader through the orders for surgical intervention, possible complications, postoperative care and diet. The text is illustrated with line drawings, which are clear and comprehensive. The book is a fine guide for everyday use in the hospital.

—M.T.

Pain, The Destroyer, and the Sympathetic Origin of Dystrophy. By Miguel Lopez Esnaurrizar. Paris: Editions Lamarie, 1959. Pp. 210, with 18 illustrations.

Everyone who wants to discover, in medical thinking, new ideas audacious in themselves and supported by an immense knowledge of living matter, will read with pleasure the monograph Prof. Miguel Lopez Esnaurrizar has written for Lamarie House of Paris. He has made the sources of knowledge understandable, with strong emphasis on his own conferences in France.

The main subject of this wise Mexican physician is the important role of the sympathetic nervous system in pathologic conditions. He has analyzed this role in its effects on internal life, following difficult clinical observations since the beginning of 1940. The facts in a surprising number of cases confirm his own conception, and show the incidence of pathologic conditions in relation to the life of the psyche. One must felicitate Prof. Esnaurrizar, who has contributed greatly to knowledge of living phenomena.

—PIERRE COMBEMALE, M.D.

Tumors and Tumorous Conditions of the Bones and Joints. By Henry L. Jaffe. Philadelphia: Lea & Febiger, 1958. Pp. 629, with 701 illustrations.

This authoritative text presents an integrated study of the tumors and tumorous conditions affecting the bones and joints. In its correlation of the data—clinical, roentgenographic and pathologic—the book is important and valuable to general surgeons, orthopedic surgeons, radiologists and pathologists alike. It considers not only the primary tumors and other tumorous conditions affecting the bones

and joints but metastatic tumors to the skeleton; tumors developing in bone at sites of damage from noxious agents, and tumors invading bone from overlying soft parts.

Careful attention is given to the diagnostic approach and the problems of differential diagnosis.

The text is concise and explicit. It covers the full range of the subject, including some types of tumors not previously presented in a text of this kind, such as *juxtacortical chondroma, juxtacortical osteogenic sarcoma, fibrous cortical defect, desmoplastic fibroma,* and *benign osteoblastoma.*

The illustrations—roentgenograms and photographs portraying the gross and microscopic changes described—are explained in detail.

The author, an acknowledged authority on his subject, gives due credit to others in the field, and their contributions are cited.

The index is thorough and the bookmaking excellent.

This text, with its fresh and contemporary view of its subject, is sound, practical, complete and informative. It is recommended to all general surgeons as well as specialists.

—MAX THOREK, M.D.†

†Deceased.

The Triumph of Surgery. By Jürgen Thorwald. Translated from the German by Richard and Clara Winston. New York: Pantheon Books, Inc., 1959. Pp. 454, with 13 illustrations.

A companion to the author's highly successful *Century of the Surgeon,* this volume carries surgery into our own time and dramatizes the work of men some of us have known.

The story begins with the Third International Medical Congress in London in 1881, which the writer sets as the date for the beginning of a new era in surgery, when men put into practice the earlier great discoveries of the nineteenth century. This panorama is seen through the eyes of Dr. Hartmann (a device also used in *Century of the Surgeon*), and the reader seems therefore to meet at first hand such men as Freud, the Mayo brothers, Charcot and many, many others. The book is based on a careful research, as was its predecessor, but is written so as to make fascinating reading for both physician and layman. In other words, it is medical history, social history and news. It should be an international bestseller.

—MAX THOREK, M.D.†

In every investigation the *search* for elementary data, often of a morphological character, preceded the search for the dynamic interrelationships among objects, i.e., *research* properly so-called. The flame has always preceded the light. So also the thirst for learning is born with human life. Aren't children always asking the how, what, and why of things? The eyes of the researcher light up with the same curiosity as a child's eyes, which look at everything in astonishment and wonder.

—*Marti-Ibañez*

Abstracts from Current Literature

The Surgical Relief of Chronic Myocardial Ischaemia. Kauntze, R., McGill, D., and Brock, R., Lancet 2:631-634, 1959.

The authors describe an operation for improving the vascularity of the myocardium. It consists of abrasion of the epicardial surfaces of the heart proper and of the visceral layer of the parietal pericardium. As an incidental step in the procedure, the left internal mammary artery is ligated.

The patients selected for operation were patients with intractable angina unresponsive to medical treatment. They were free of cardiac failure, severe hypertension and generalized arterial disease. Ten cases are reported in which the patients have been followed for six months or longer. The authors state that they have a number of additional patients whom they are following at present, but on whom, with less than six months of follow-up, they do not consider it justifiable to report.

Their results were considered excellent in 3 instances, good in 5 and poor in 1. One patient died as a result of the operation.

Postoperative evaluation was principally on a clinical and subjective basis.

—CHURCH E. MURDOCK, JR., M.D.

Early Diagnosis of Multiple Pregnancy. Novotny, C. A., Haas, W. K., and Callagen, D. A., J.A.M.A. 171:116-880. 1959.

The authors have found in this study that it is possible to make an accurate diagnosis of single or multiple pregnancy or to confirm the occurrence of fetal death in the vast majority of all patients studied. Ninety per cent accuracy of diagnosis was achieved between the twentieth and the twenty-seventh week, the most favorable period for application of the test. The test is the Grass electroencephalographic procedure. One hundred per cent accuracy of diagnosis was obtained also in cases of multiple pregnancy during this same period.

The test is easily performed by a technician trained in electroencephalography and, as used in the Naval Hospital, does not require the presence of a physician during the recording. Because of its ease of application, accuracy and safety, as well as its overwhelming acceptance by patients, the U. S. Naval Hospital, Portsmouth, Virginia, now employs the test as a part of the prenatal routine in all cases of suspected multiple gestation or of intrauterine fetal death.

—EDMUND LISSACK, M.D.

The Condition of the Vagina After Vaginal Radical Operation. Scholz, H., Zntrlbl. f. Gynäk. 80:83, 1958.

Since 1945 a vaginal radical hysterectomy (Stoeckel's technic) had been performed on 500 patients. Prior to removal of the uterus and parametrium a rather large vaginal cuff was built, followed by an extensive Schuchardt incision on the left side. Fixation of the vaginal edges to the levator or to the round ligament stumps was not done. In cases with considerable prolapse of the bladder a tobacco-pouch suture or several interrupted sutures were used for repair.

A series of 164 patients, averaging 49.8 years of age, who had been operated on within the past ten years and who had not suffered a recurrence, were available for follow-up, which established these facts:

1. Total prolapse of the vaginal stump occurred in 3 per cent.
2. There was vaginal descent in 67 per cent, involving the anterior or posterior wall in 44 per cent and both walls in 23 per cent.
3. There was descensus of the anterior wall in 32.9 per cent, with or without a rectocele. In 19.5 per cent the descent was slight, in 9.2 per cent moderate and in 4.2 per cent severe.
4. In 24.4 per cent there was relative or absolute incontinence of urine, very slight in 11.6 per cent and considerable in 12.8 per cent. Of all the patients with incontinence, 42.5 per cent had, in addition, anterior vaginal descensus, whereas 57.5 per cent did not.

5. In 57.3 per cent there was descent of the posterior wall, with or without a cystocele. Of these, 35.9 per cent had a small, 15.3 per cent a medium and 6.1 per cent a large rectocele.

6. The average number of confinements had been 1.7. The patients with vaginal descensus, however, had had an average of 1.9 confinements, and those with incontinence 2.1.

7. Of all followed-up patients, 17 per cent were nulliparae.

8. Of the patients with vaginal descent 14.5 per cent were nulliparae as were 7.5 per cent of those with incontinence.

These data point to the necessity of performing a plastic operation on the vaginal walls and bladder sphincter in connection with vaginal radical hysterectomy. It is notable that in more than half of the patients with incontinence of urine no descensus of the anterior vaginal wall could be observed, and that descensus of the posterior wall was much more frequent.

The normal length of the vagina is 7 to 10 cm. Measurements during follow-up examinations of the 164 patients revealed the length of the vaginal stump to be 7 cm. or longer in 26.8 per cent; 5 to 7 cm. in 41.5 per cent; 3 to 5 cm. in 28 per cent, and less than 3 cm. in 3.7 per cent. Consequently, one may assume that shortening of the vagina does not occur more often after vaginal than after abdominal panhysterectomy.

Finally, it was observed that the mechanical possibility of cohabitation was retained in 50 per cent of the cases and had become difficult or impossible in only 8 per cent as a result of radical vaginal intervention.

—ERNEST G. ABRAHAM, M.D.

Treatment of Melanoma by Isolation-Perfusion Technique. Creech, O., Ryan, R. F., and Krementz, E. T., J.A.M.A. **169**:339, 1949.

The authors have perfused carcinocidal drugs into isolated tumor-bearing areas using an extracorporeal circulatory system consisting of a pump and a disposable oxygenator. Suitable plastic cannulas are inserted into the artery and vein of the extremity. An Esmarch bandage is placed at the crotch and pulled tightly to obstruct superficial flow. The blood is oxygenated with 100 per cent oxygen at a flow rate of 5 L. per minute. After venous outflow and arterial inflow have been balanced, phenylalanine mustard is injected into the arterial line. On completion of perfusion the venous line is disconnected and the area drained of venous blood. Five hundred cc. of fresh blood is injected through the arterial cannula and removed via the venous channel. The cannulas are removed, the openings closed and the tourniquet released. Leukocyte and platelet counts are checked at intervals in order to determine the effect of hematopoiesis. The results in this series were encouraging; regression and even disappearance were noted.

—ERNEST G. DEBAKEY, M.D.

Surgical Management of Thyroid Cancer. Thomas, C. G. Jr., Southern M. J. **52**:260, 1959.

The author has reviewed the natural history of carcinoma of the thyroid and classifies such tumors into: (1) poorly differentiated or solid carcinoma, (2) papillary adenocarcinoma, and (3) alveolar adenocarcinoma.

Ten years after diagnosis the mortality figures are as follows:

Poorly differentiated tumors..80 per cent
Papillary carcinoma .....60 per cent
Alveolar carcinoma .....20 per cent

He stresses the fact that in the cases of papillary and alveolar neoplasm there is a relation to thyrotropin. This relation suggests that thyroid-stimulating hormone may serve as a promoting factor in the origin of these tumors. In the author's opinion the ideal surgical approach to a lesion of this type is unilateral lobectomy as a method of biopsy. If the tumor is completely surrounded by normal glandular tissue and if there is no gross evidence of spread, the operation is complete, and then replacement therapy with thyroid hormone is considered in the subsequent management of the patient. There is a place for incisional biopsy, especially to differentiate between diffusely infiltrating carcinoma and diffuse thyroiditis. He concludes that a nearly total thyroidectomy, the posterior capsule being left on the normal side, will diminish the incidence of hypoparathyroidism in those cases

in which total thyroidectomy is contemplated. The ultimate criterion for neck dissection in cases of thyroid carcinoma is the presence of enlarged lymph nodes, as determined by the operative observations. Anatomically, lymphatics of the neck are described in detail. When postoperative hormonal regulation is required or indicated, desiccated thyroid has the advantage of low cost and ready availability for long-term management.

—JAMES H. ERWIN, M.D.

Pancreaticoduodenal Wounds (Les Plaies Duodeno-pancreatiques). Delom, Mattei and Hervé, J. de Chirurgie 75:248, 1958.

Seventeen combined pancreatic and duodenal wounds were discovered during the exploration of 675 abdominal war wounds. In 16 cases the lesions were associated with important injury to neighboring organs. Of the 17 patients, only 8 recovered. Recovery is frequently associated with chronic pancreatitis and diabetes.

Wounds in this area are serious because of the anatomic arrangement of the duodenum and the pancreas, which are surrounded by such important vessels as the superior mesenteric artery and vein, the portal vein, the splenic pedicle, the celiac trunk, the transverse mesocolon, etc. Injury to the celiac plexus may cause intense shock. The bile ducts and pancreatic ducts are easily injured, and fistula formation results. The pancreas is friable, and autodigestion occurs with injury. Posterior wounds of the duodenum are difficult to detect at operation but should be suspected when bile staining or retroperitoneal hematoma is observed.

Large ragged wounds of the pancreas should be packed. Removal of the tail of the pancreas may be needed, with concomitant splenectomy. Blood loss may range from 300 to 1,900 cc.; antibiotics should be used to prevent infection of the ever-present hematoma. The use of morphine, intravenous dextrose and amino acids should be avoided, as they stimulate pancreatic secretion. Abscesses should be drained. Pseudocysts should be marsupialized or drained. Associated hepatic injuries are common and occurred in 8 cases. Lesions of the gallbladder occurred in 3; of the spleen in 2; of the stomach in 6; of the small intestine in 2, and of the transverse mesocolon in 7.

The direction of the entrance wound should arouse suspicion. Roentgenograms showing foreign bodies close to the liver may point to pancreatic injury.

The gastrocolic omentum should be opened. The pancreas must be checked under direct vision.

Lumbar lordosis should be exaggerated to increase the exposure. The hepatic flexure of the colon should be mobilized and the liver and stomach retracted upward. Intestinal wounds should be sutured without tension.

The stomach should be aspirated and a duodenal tube left in place. A bypass gastroenterostomy may be used with duodenal wounds, to diminish the tension on suture lines there, or where duodenal continuity can not be reestablished. A loop of jejunum may be anastomosed to large holes in the third part of the duodenum. Various maneuvers may be utilized on the basis of these principles.

All wounds of the pancreatic parenchyma must be sutured to stop bleeding. The pancreatic area must be drained. The risk of removing a foreign body in the area of the pancreatic and biliary ducts should be measured against the risk of leaving it.

When the duct of Wirsung is divided, it may be necessary merely to ligate both ends, drain and pack, as the patient may not be in shape for a partial pancreatectomy. Any injury to the spleen is an indication for splenectomy, Shock must be treated in the usual way, by rapid blood transfusion. In some of the authors' cases procaine hydrochloride was injected about the celiac plexus.

—J. A. MCLENNAN, M.D.

An Evaluation of the Eleventh Intercostal Space Incision for Renal Surgery. Presman, D., J. Urol. 82:18-20, 1959.

The author describes his technic in approaching the kidney. The incision is made between the eleventh and twelfth ribs. By carefully cutting the diaphragmatic muscle slips at their insertion on the twelfth rib, opening of the pleura can be avoided.

One of the advantages of this incision is that direct visualization of the upper pole is

obtained. Another is that it is easier to apply pedicle clamps, because of the higher level of the posterior portion of the incision. Additional exposure may be obtained if necessary by resection of the eleventh rib. If necessary, the diaphragm may be opened for a transthoracic approach.

When patients have undergone previous renal operations by the standard subcostal approach, the approach to the kidney can be made easier by the technic aforedescribed, since the incision avoids the dense scar tissue from the earlier operation.

—SHEPARD JEROME, M.D.

Pathologic Implication of the Catechol Amines, Epinephrine and Norepinephrine. Szakacs, J. E., Dimmette, R. M., and Cowart, E. C., U. S. Armed Forces M. J. 10:908, 1959.

Earlier investigators have recognized both the local and systemic lesions produced by epinephrine. Norepinephrine similarly has tissue-necrotizing properties, and its local complications are common knowledge. The systemic lesions produced by norepinephrine were recognized only recently, although for years Raab emphasized the fact that knowledge of the significance of the pathogenesis of these lesions is far from complete.

At least two different mechanisms are responsible for the pathologic changes produced by norepinephrine. One is mechanical and due to hemodynamic factors; the other is a chemical factor producing derangement in cellular oxidation by uncoupling oxidation from phosphorylation. The best examples of the first mechanism are the massive cerebral hemorrhage and hemorrhage into the mitral valve. These lesions depend more on the rate of the infusion than on the total dose administered, and can be produced by a single injection. The second mechanism is best illustrated by the myocardial necrosis following low dosage and constant rate infusions of norepinephrine. It is not associated with marked changes in blood pressure or with valvular hemorrhage. Hemodynamic changes then are combined with the hypoxiating effects of norepinephrine in the production of lesions of the liver, kidneys and large vessels. Additional factors operate in the production of skin necroses. These factors include the hydrostatic pressure of the infiltrating fluid and local anatomic peculiarities of the vessels, as in the case of the lower extremity.

Norepinephrine, while it is truly a life-saving drug when used as a specific therapeutic agent in the postoperative management of pheochromocytoma, is a pathogen, and nonspecific indications for its use should be critically evaluated.

WILLIAM E. NORTH, M.D.

Surgery of Neoplasms of the Prancreas (Cirugia De Las Neoplasias Del Pancreas). Estrada, L., Cir. Gin. Urol. 13:187, 1959.

The credit for experimental work on pancreatic surgery dates back to Johan Brunner in 1673. Surgical treatment of the pancreas in recent times, however, is linked with the name of Whipple.

Primary pancreatic carcinoma takes many forms—diffused, localized, infiltrating, smooth and irregular. It may occur in the head of the pancreas as well as in the body or the tail. The former is probably the more frequent site.

Adjacent organs are usually involved, although metastasis is frequent. The involvement of related organs usually results in the presenting symptom complex of jaundice, gallbladder disease and the like. Pain is the most characteristic subjective sign. Objectively, a palpable tumor in the epigastrium, with hepatomegaly, splenomegaly, ascites and digestive hemorrhage, is not infrequently demonstrable.

—JOHN A. ZIEMAN, M.D.

IMPORTANT ANNOUNCEMENT

Contributors of scientific articles for publication in The Journal of the International College of Surgeons from Europe, the Near East and the Middle East should send their articles to:

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Un comité a été nommé pour l'examen des articles à paraître. Les auteurs sont priés de joindre à leur travail de brefs résumés en français, anglais, allemand, espagnol, italien et portugais si possible.

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